Formerly

ROYAL SANITARY INSTITUTE

FOUNDED 1876

TO PROMOTE THE HEALTH OF THE PEOPLE

LIBRARY REGULATIONS

(a) Books, periodicals and pamphlets may be borrowed by Fellows, Ordinary Members and Associates personally or by a messenger producing a written order. The person to whom such publications are delivered shall sign a receipt for them in a book provided for that purpose

(b) Publications may be borrowed through the post, or by other means of carriage, upon a written order. The postage or carriage of publications returned to the Institute shall be defrayed by the borrower.

(c) A borrower may not have more than three publications in his possession at one time.

(d) A borrower will be considered liable for the value of any publication lost or damaged while on loan to him, and, if it be a single volume or part of a set, for the value of the whole work thereby rendered imperfect. Marking or writing in the publications is not permitted, and borrowers are requested to call attention to damage of this character.

(e) Books and pamphlets may be retained for twenty-eight days. Periodicals may be retained for fourteen days. Applications for extension of the loan period must be made in writing before its expiry. No publication may be kept longer than three months.

(f) Books and pamphlets added to the library will not be lent until after the expiry of one month from the date received. The current number of a periodical may not be borrowed.

(g) Borrowers retaining publications longer than the time specified, and neglecting to return them when demanded, forfeit the right to borrow until they be returned, and for such further time as may be ordered.

Any borrower failing to comply with a request for the return of a publication shall be considered liable for the cost of replacing it, and the Council may, after giving due notice to him, order it to be replaced at his expense.

No publication may be reissued to the same borrower until at least seven days have elapsed after its return, neither may it be transferred by one borrower to another.

(h) Publications may not be taken or sent out of the United Kingdom.

(i) Publications returned through the post must be securely packed in a box or otherwise adequately protected.

(j) The Library may be used for reference by Fellows, Ordinary Members and Associates during the office hours of the Institute.

(k) Parcels should be addressed :

THE ROYAL ~~SANITARY INSTITUTE,~~ Society of Health
90, BUCKINGHAM PALACE ROAD,
LONDON, S.W.1.

1000/52

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

JANUARY 1952 Vol. 11

CONTENTS

SECTION I.—GENERAL

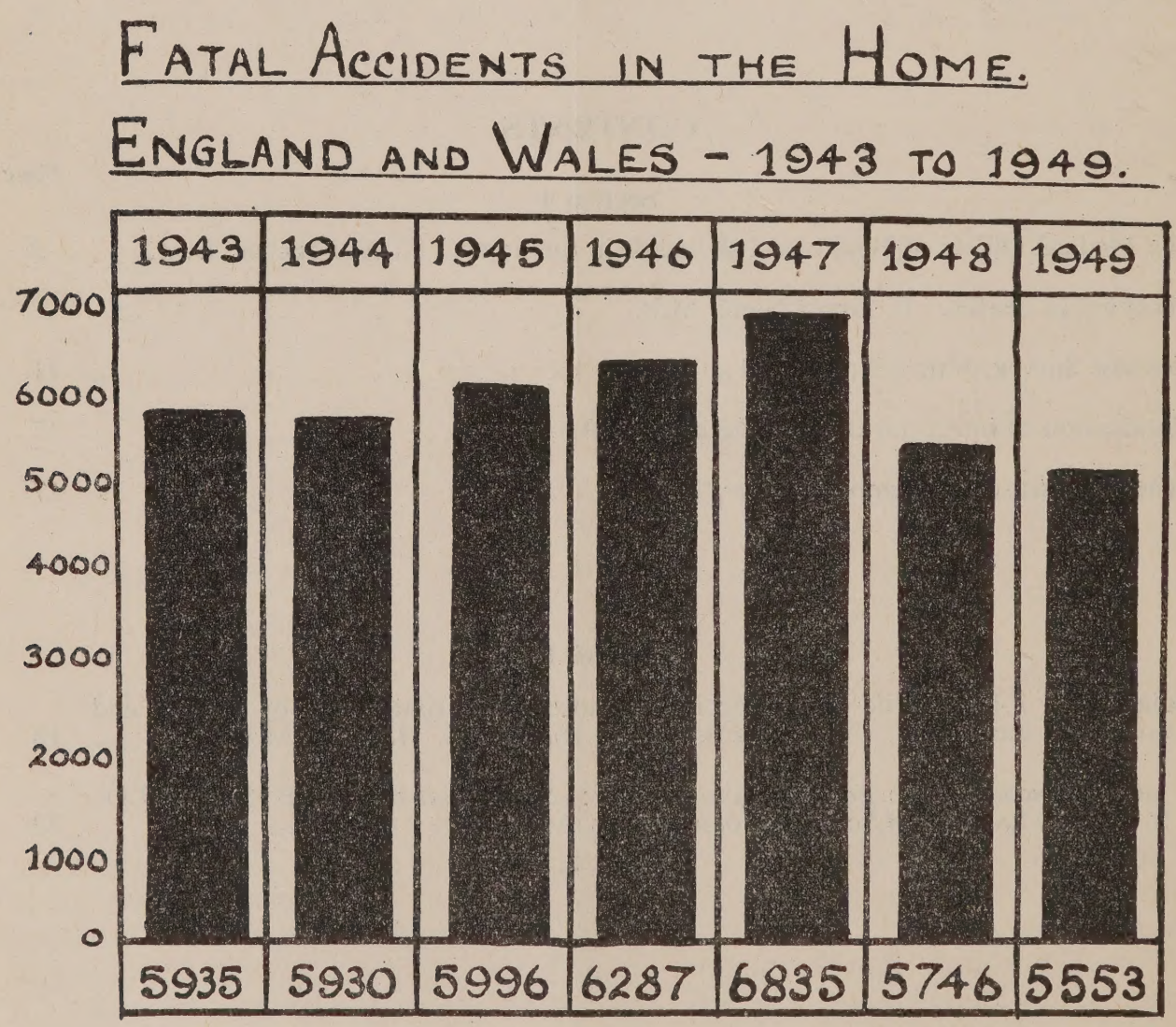
Issued from the Office of the Ministry of Health, Savile Row, W.1

THE MEDICAL OFFICER OF HEALTH AND ACCIDENTS IN THE HOME

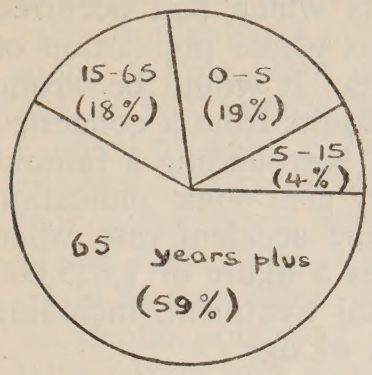
C. A. Boucher, D.M., D.P.H., Ministry of Health

In 1946 the Chief Medical Officer of the Ministry of Health in his report "On the State of the Public Health during Six Years of War" observed that "One cause of mortality, and therefore presumably of morbidity as well, that has not received the attention it merits, is accidents in the home". In the following year the Home Secretary set up a Standing Interdepartmental Committee on accidents in the Home, which was to co-ordinate actions by the various departments concerned with domestic accidents and maintain contact with voluntary and other unofficial organisations interested in the problem.

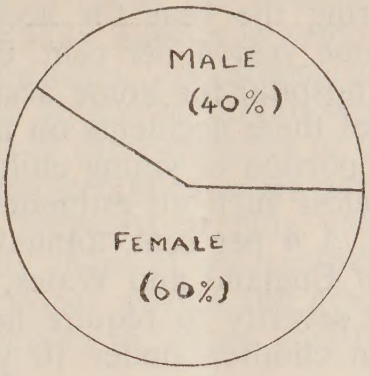
In England and Wales about 6,000 persons die every year as a result of accidents in their homes, as the following chart will show; 1949, the last year for which figures have been made available to the Committee, shows a total of 5,553 deaths which is a distinct improvement on previous years.



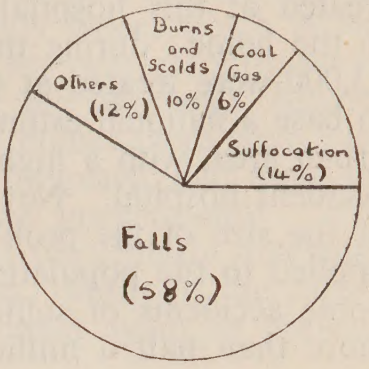
The following diagrams give further information about these fatal domestic accidents:—



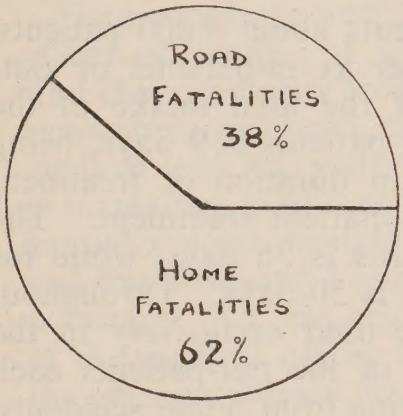
Age groups



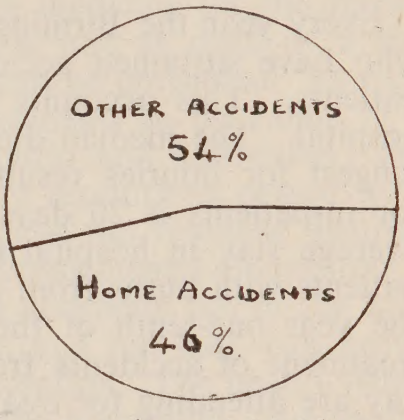
Sex distribution



Types of fatality



Fatal home compared with fatal road accidents: children under 15 years.



Proportion of fatal home accidents to fatal accidents from all causes.

From these diagrams and other sources the following information is now obtained:—

1. There has been no appreciable change in recent years in the number of fatal home accidents in England and Wales.

2. Four-fifths of these fatalities occur in young children under 5 years and in elderly people over 65 years of age.

3. Three-fifths of the fatalities strike the female sex, but this is mainly due to the large number of fatalities in elderly women; more male than female children under 15 years are killed in their own homes.

4. *More children under 15 years are killed in their own homes than die from accidental causes elsewhere, including road accidents.*

5. *More children under 15 years die from accidents in the home than die from any single infectious disease.*

6. In children between 1 to 5 years a fatal home accident is the third largest cause of death.

Serious Non-fatal Home Accidents

Accidents in the home are not notifiable and there is thus no exact information for England and Wales, but a reasonable estimate can be made from figures available in certain parts of the country. In 1946 it was found at the Edinburgh Royal Infirmary that of all the accidents treated in the out-patients department 23 per cent. occurred in the home. In 1946 a careful survey was made of the area, with 13,000 inhabitants, surrounding

the Birmingham Accident Hospital; it was found that 9 per cent. of the children under 10 years and 3 per cent. of older children and adults were treated at this hospital during the year for accidents which had occurred in the home; during the same year 4 per cent. of this whole population of 13,000 were treated at this hospital for home accidents. It would be wrong to base a national estimate of these accidents on the results from a congested urban area, with a high proportion of young children, surrounding a famous accident hospital. Nevertheless such an estimate may give some indication of the size of the problem; a 4 per cent. annual home accident rate, when applied to the population of England and Wales, gives a figure of 1,675,000 home accidents of sufficient severity to require hospital treatment, including more than half a million in children under 10 years of age.

Economics of Home Accidents

Every year the Birmingham Accident Hospital treats about 5,000 patients, who have sustained accidents in their homes, either as in-patients or out-patients. This accounts for about 18 per cent. of the total intake of the hospital. The median duration of treatment for out-patients is 9 days, being longest for injuries resulting from falls: the median duration of treatment for in-patients is 20 days, followed by 17 days out-patient treatment. The average stay in hospital for patients with home burns is 35 days, while for patients with burns from clothing catching on fire it is 50 days. Throughout the year one-tenth of the beds of this hospital are used *exclusively* in the treatment of accidents from the home. About 60 of the out-patients each day are attending for treatment of the injuries resulting from home accidents.

For the year 1949 the estimated overall cost of the treatment of domestic accidents at this hospital was £52,000, and it would probably be accepted that the high standard of treatment at this hospital probably produces quicker results than at the average hospital. Adjusting this treatment cost to a national scale it is estimated that the annual national figure for the treatment of accidents arising in the home would be £4–£5 million; and costs have been rising since 1949. It is estimated that the cost of in-patient treatment for burns and scalds alone, arising from accidents in homes in England and Wales, amounts to nearly £1 million.

These figures take no account of the indirect cost to the community. Industry will suffer because the affected worker is absent, or he or she may be absent to care for an injured child or elderly relative at home; the injured child may lose long periods from his school. The general practitioner will spend time in the treatment of these accidents; while the local authority may have to divert health visitors, district nurses, home helps and welfare officers to such cases. Sickness benefit may have to be provided, and financial relief may be sought from the National Assistance Board. Nor do these factors take into account the personal tragedy associated with so many of these accidents.

Are Home Accidents Preventable?

In various parts of the country there have been surveys into home accidents; these have shown that structural defects in the homes could be responsible for one-third of the cases, and that in half of these the defects resulted from lack of maintenance. Most of the remainder are considered to result from ignorance of the dangers that exist and lack of reasonable care. Sixty-seven per cent. of the scalds treated at the Birmingham Accident Hospital were in children under five years, and half of these were considered to be easily preventable with ordinary care: in 90 per cent. of the burning

accidents from coal, gas or electric fires no fire-guard was in use at the time of the accident, and in most of the remainder the guard was not attached to the fire and had been removed by the victim. Experience shows that these such accidents do not normally affect the same family twice, which is testimony to one form of education. A fireguard survey was recently carried out in 987 homes by the Regional Organisers of the Women's Advisory Council on Solid Fuel, and in three-quarters of these homes there were young children: 23 per cent. of these homes never used a fireguard, and more than half either never used a fireguard or only used one intermittently. Many of the fatal falls, especially those of old people and particularly downstairs, could be prevented with proper advice and attention. Many of the cases of accidental poisoning, especially in young children, could again be prevented with advice and education.

The Role of the Medical Officer of Health

Here is a large and satisfying field of preventive medicine for the medical officer of health. A child who dies from burns is just as dead as a child who dies needlessly from diphtheria : both deaths can be prevented. In recent years there has been increasing publicity about accidents in the home in the medical and lay journals, in the Press, on the Radio and Television; there is increasing public concern as is demonstrated by resolutions passed at the annual general meetings of responsible bodies such as the Womens' Institutes and the Womens' Voluntary Services. The Interdepartmental Committee are convinced that the key to the prevention of these accidents lies in the hands of the medical officer of health because he alone, and his staff, can provide the personal advice that will be listened to with attention. The health visitor's field is now widened to include all members of the household; the sanitary inspector has certain statutory rights of entry to any household: the district nurses, home helps and others have valuable parts to play.

Under existing legislation the medical officer of health has full powers to undertake this work:—

National Health Service Act, 1946. Section 28. "A local health authority may with the approval of the Minister, and to such extent as the Minister may direct shall, make arrangements for the purpose of the prevention of illness, . . ." "A local health authority may, with the approval of the Minister, contribute to any voluntary organisation formed for any such purpose as aforesaid".

Local Government Act, 1948. Section 136. "A local authority in England or Wales may, with the consent of the Minister given either generally or specially, contribute towards the expenses of any body carrying on activities within the area of that authority, . . . or of giving advice, information or other assistance to persons resident therein, or otherwise for the benefit of that area or those persons".

Possible Methods of Approach

1. The medical officer of health of the local health authority could initiate a survey of his area, and this might well be done by one or more of his health visitor staff; such a survey would not only provide valuable information about the morbidity of home accidents but would also enable personal education and advice to be given in the homes.

2. These home accidents are not legally notifiable but the medical officer of health could by his influence get information from the local hospitals, or hospital management committee, and the general practitioners, from which information he could devise the most suitable preventive measures.

3. The medical officer of health could draw attention to this problem in his annual report, and thus stimulate the interest of his public.

4. He could initiate local action by drawing the attention of his health committee and council to this problem.

5. He could gain wider publicity in this respect by the use of the local Press.

6. The medical officer of health of the local health authority could arrange for the education of mothers on this subject at the maternity and child welfare clinics: he could perhaps arrange for teaching to be given to older school-children, particularly girls; and for advice to be given to old people through the local welfare associations. He could offer to provide speakers for the women's and other social organisations; a close link with the St. John Ambulance Association and the British Red Cross would be of great value.

7. He could ask the Coroner to give him information about fatal home accidents, and he could then follow these up, when thought desirable.

8. The Interdepartmental Committee considers that the Home Safety Department of the Royal Society for the Prevention of Accidents should play a most valuable part in the prevention of accidents, and that greater use should be made of its services by local authorities. Under the legislation mentioned above a local health authority can contribute to this organisation: in return it would receive Home Safety posters, "Safety News", the "Home Safety Bulletin", leaflets and pamphlets, and lecturers. Similarly a local authority could establish a local home safety committee to which would be made available all the facilities described above; 33 successful home safety committees have been established in England and Wales. Enquiries should be made to The Home Safety Department, Royal Society for the Prevention of Accidents, Terminal House, Grosvenor Gardens, London, S.W.1.

9. Trailer films on home accidents have been appearing in the commercial cinemas since 1947, but are not available for showing by local authorities. The following films can, however, be obtained by them:—

"Playing with Fire" (11 minutes). Borrowers having a 16 mm. sound projector can obtain this from the Central Film Library, Government Building, Bromyard Avenue, Acton, London, W.3. Borrowers not owning a projector should apply to their Regional Film Officer of the Central Office of Information (or to their headquarters at 83, Baker Street, London, W.1).

"Fiery Accidents" (1 minute). Can be shown in cinemas, with the agreement of the management. Application to National Screen Service Ltd., Wallace House, 113, Wardour Street, London, W.1. No charge.

"Dangerous Ages" (4 minutes), "The Human Factor" (14 minutes), "Who stated it?" (11 minutes). These 16 mm films can be obtained from the Royal Society for the Prevention of Accidents.

10. A display set, "You and your baby", is available to the medical officer of health. It is suitable for maternity and child welfare clinics and consists of twelve upright panels, $12'' \times 20''$: one panel is devoted to the prevention of burns and scalds, including advice on guarding of fires and the danger of flannelette nightgowns near unguarded fires. Application to the Circulation Section, Publications Division, Central Office of Information, Block 2, Montagu Mansions, Crawford Street, London, W.1. No charge.

11. Advice notes on home accidents, which are circulated to the Press from the Ministry of Health, can be obtained from Mrs. Samson, Ministry of Health, Savile Row, London, W.1.

12. Pictorial pamphlets and leaflets can be obtained from the Royal Society for the Prevention of Accidents. They would serve a useful purpose in maternity and child welfare clinics, school medical clinics, waiting rooms of general practitioners and hospital out-patient departments.

13. The medical officer of health may wish to arrange a home safety exhibition, which should attract wide interest. A useful addition to this exhibition will be " Trouble House ", which shows a home with accidents; it was displayed at the Ideal Home Exhibition 1951 and can now be obtained from the Royal Society for the Prevention of Accidents for a hiring charge of £10 per week, plus transport charges. " Accidents in the Home ", a three panel display set, can be hired at 10s. a week from the Exhibitions Officer, Central Council for Health Education, Tavistock House, London, W.C.1.

The Crusader Insurance Company own a home accident display set for local home safety exhibitions or for local clinics: they also own a smaller portable set. Both are available free of charge. Applications to Miss I. H. Charley, S.R.N., Crusader Insurance Company Ltd., 14, Pall Mall, London, S.W.1.

14. The medical officer of health of the district authority can give special attention to the design and equipment of new houses with special reference to the safety aspect. Reference to this matter is made in paragraph 175 of the Housing Manual 1949. Advice could be given on adequate natural and artificial lighting and the design, for safety purposes, of the kitchens. There are now British Standards specifications for guards for gas and electric fires, and the medical officer could ensure that fires in local authority houses be fitted with such guards. He could also consider a scheme of hiring fire-guards similar to that practised by the Edinburgh Corporation.

Conclusions

It is considered that the human factor is responsible for a large proportion of these accidents in the home, that the underlying cause is often ignorance sometimes associated with carelessness, and that mothers and older schoolchildren are the best groups to educate on this subject. The medical officer of health is the person best suited to carry out the preventive and advisory work in this field, which can prove of absorbing interest; he and his staff have a personal and intimate interest in the health and welfare of the local people, and the people would respond to his advice.

In 1624 Sir Henry Wotton published his " Elements of Architecture ". He says, regarding staircases, " They should have a very liberal light, against all Casualty of Slips and Falls ".

In the " Gentleman's Magazine " of 1803 appeared the following, " One of the most evident methods to prevent the cloaths from catching fire is to have wire fenders placed before the fireplace, of a sufficient height to hinder the coals from flying into the room . . . the safest are those of close wirework . . . and may we not attribute many of the melancholy events, which have happened of late, to the prevailing custom of wearing muslin dresses? "

Three hundred years after the book was published houses are still being built with badly lit staircases. One hundred and fifty years after the issue of the magazine we are still deploring the absence of fireguards and the highly inflammable nature of flannelette and wynceyette material.

A leading article in the " Medical Officer " of 3rd November, 1951, says, " Medical officers of health have not contributed a great deal to the sum of available knowledge about the causes, course and effects of accidents in the home, for all that in our health visitors we have a means of discovering much more than observers whose base of operations is in the hospital, or even the general practitioners. We can complete much more fully than they can hope to do the picture of total morbidity from this cause, . . .". Is not this a challenge?

ENCEPHALITIS DEATHS

C. Grant Nicol, M.B., D.P.H., Barrister-at-law, Ministry of Health

The subject of acute encephalitis is much discussed at the present time, but the number of deaths certified to be due to it is comparatively small. It seemed, therefore, that an analysis of the clinical history and post-mortem findings in fatal cases of "encephalitis" reported during a year might throw useful light on its aetiology. Through the courtesy of the Registrar-General arrangements were made whereby each month copies of the essential information on all certificates giving "encephalitis" as a cause of death were sent to the epidemiological section of the Ministry of Health. A covering letter, explaining that help was desired in an investigation being undertaken into the aetiology of encephalitis, was prepared and a list of headings enclosed with it indicating those aspects to which the inquiry was directed. These included "duration and nature of illness with date of onset", "brief clinical history", "date of death", "previous illnesses", "inoculations during the year before onset", "post-mortem findings", "history of illness in the family or other close contacts" and an opinion as to whether the illness should be regarded as due to a communicable disease, to an infection or to a degenerative condition.

These (the letter and list) were sent to the medical superintendent, secretary or other appropriate officer in cases where the death had occurred in and was certified from a hospital; to the practitioner, where the death had occurred in his practice; and to the coroner where the death had been reported to him for further investigation. Many full replies were received each month and an analysis of the information obtained during the 12 months July, 1950, to June, 1951, may be of interest.

Deaths, which could be regarded as due to one or other of the forms of encephalitis, were coded in accordance with the "Manual of the International Statistical classification of diseases, injuries and causes of death" (1948). This is not the place, however, to discuss the implications of this complex method of coding. The number of deaths certified as due to "encephalitis" was 255.

The certificate was accepted without inquiry in 14 instances, and inquiry was made in 241. To these 241 inquiries, 175 replies were received: in 66 instances no reply was received.

In 14 cases the reply showed that the death was due to chronic encephalitis or to some other condition outside the inquiry.

The results of the inquiry into 241 deaths from "acute encephalitis" are summarised in the table below. (Table I.)

Of the 175 deaths, about which replies had been received to the inquiries, or the certificate had been accepted without inquiry, 136 appear to have been due to acute encephalitis of one or other form, and 39 deaths were ascribed to other causes.

Of the 136 deaths above, 43 were due to or associated with infectious disease, generally measles (Table II). Eleven of the "confirmed" encephalitis deaths were on inquiry assigned to polioencephalitis.

Of the original 175 replies, therefore, 39 were "not confirmed", but were due to other causes; 43 were "post infectious"; and 11 were "polioencephalitis", leaving 82 "confirmed" deaths from acute encephalitis. One or two interesting matters emerged from the inquiry: in one infant death

I.—*Deaths assigned on death certificate to "Acute Encephalitis" by age group*

Age Group	All deaths so assigned	Number in which this diagnosis was confirmed	On findings respectively of			
			Post-mortem and histopathology	Post-mortem macroscopic only	Clinical and laboratory investigations	Clinical signs only
Under 1 year	34	17	—	14	1	2
1–4 ...	65	43	3	32	3	5
5–9 ...	25	18	1	7	4	6
10–14 ...	6	4	—	2	—	2
15–24 ...	20	10	2	8	—	—
25–34 ...	19	13	2	11	—	—
35–44 ...	19	11	4	6	1	—
45–54 ...	22	10	1	8	—	1
55–64 ...	15	4	—	3	1	—
65+ ...	16	6	1	2	1	2
All ages ...	241	136	14	93	11	18

Note:

In 39 cases the diagnosis of acute encephalitis was negatived; in 14, by histopathological examination; in 20, by macroscopic appearances at post-mortem and in 5, by further information. The age distribution of these 39 deaths due to other diseases showed a fairly uniform spread. The revised diagnoses in these 39 cases were:—in 10, polioencephalitis; in 17, conditions closely allied to acute encephalitis; and in 22, conditions only superficially resembling encephalitis. The 10 patients who died of polioencephalitis were all under 30 years of age.

No reply was received to the inquiry in 66 instances.

II.—*The age distribution of deaths from acute encephalitis associated with Infectious Diseases*

Age Group	Total Deaths	Infectious Disease associated		
		Measles	Influenza	Other
Under 1 year ...	5	5	—	—
1–4	25	21	1	2 W. Cough; 1 Parotitis
5–9	17	17	—	—
10–14	0	—	—	—
15–24	3	—	2	1 virus pneumonia
25–34	5	1	4	—
35–44	4	1	2	1 epidemic hepatitis
45–54	2	—	1	1 parotitis
55–64	0	—	—	—
65+	2	—	2	—
All ages... ...	63	45	12	2 Wh. Cough; 2 parotitis; 1 virus pneumonia; 1 hepatitis.

Note.—Of these 63 deaths, there were 20 as to which no replies were received. In 9 of these (8 of measles, one of whooping cough) the certificate however, stated that the diagnosis was supported by post-mortem evidence; in 2 (both measles) by laboratory tests and clinical evidence. 17 of the 20 were associated with measles and 18 were of children under 8 years of age.

the question of death from mercurial poisoning from teething powders was raised; in one woman's death, poisoning from arsenical pessaries "could not be excluded"; another woman's death was ultimately ascribed to lead poisoning.

In only 28 cases was the post-mortem examination followed by a histological examination of the brain, and of these 28 deaths, 14 were confirmed as being due to encephalitis and 14 were disproved—in some cases disproof by a histological examination followed an earlier "confirmation" by a macroscopic post-mortem examination. In only one instance was death originally ascribed to an inoculation, and this ascription was disproved by neuro-histology, the appearances of encephalitis being due to venous thrombosis.

The terminal illness in so many of these patients followed such a closely similar course in both "confirmed" and "unconfirmed" cases that it would appear to be extremely difficult to make a correct diagnosis of encephalitis as a cause of death, without the fullest laboratory and neuro-pathological investigations. Pyrexia, headache coma, convulsions and various neurological manifestations were common to nearly all these patients' clinical histories. The real causes of death after such an illness were diverse as the selection below of ultimate findings indicates:—

In *infancy,* cerebral anoxaemia associated with the dehydration of intestinal infection, and convulsions associated with pneumonia. At *all ages,* cerebrovascular disease—venous thrombosis, embolism from mitral valve, hypertensive encephalopathy and cerebral tumour; virus encephalitis, polioencephalitis, post-infectious demyelination, other forms of demyelination disease, and, in one instance, torulosis of the central nervous system.

The subject of deaths certified as due to "acute encephalitis" appears to be worthy of further study.

SURVEY OF SICKNESS—JUNE QUARTER, 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

The Registrar General's Quarterly Return No. 411 (in Tables A to H) gives detailed results of the Social Survey's interviews of random samples of the adult population, carried out during May to August, 1951, in which the experience of April to June was recorded. The report given here presents some rates derived from these tables without standardization or correction for the number of days in the month, and compares them with those for preceding months. More detailed studies appear from time to time in the publications of the General Register Office.

Since the beginning of 1951 results from the Survey of Sickness have related only to persons aged 21 and over, not 16 and over as formerly.

The "Monthly Health Index"—the proportion of persons per 100 interviewed who suffered no illness or injury, so far as they remembered, during the stated month—is given in Table A, separately for men and for women, at ages 21-64 and ages 65 and over. In none of the four groups did the health index during April to June, 1951, show much difference from the corresponding months of 1949 and 1950.

Table B gives monthly illness rates, the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only, distinguishing persons with illness commencing in the month from those with illness continued from the previous month. The table also gives days of incapacity reported in each month and numbers of medical consultations. Including persons with an injury the average rates for the June Quarters of 1949, 1950, and 1951 compare thus:—

	Ages 21–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
Apr.–June, 1949 ...	67	80	40	83	159	65
Apr.–June, 1950 ...	66	82	43	85	137	76
Apr.–June, 1951 ...	66	76	42	84	118	70
Apr.–June 1951 per cent. of Apr.–June						
1949	99	95	105	101	74	108
1950	100	93	98	99	86	92

At both ages the level of sickness during these three June Quarters remained practically unchanged. Incapacity in April-June, 1951, was rather less than in April-June, 1949 and 1950, at ages 21-64, and considerably less at ages 65 and over. At both ages the medical consultation rate in April-June, 1951, was a little higher than in the corresponding period of 1949 but less than in 1950.

Table B also shows that during the separate months April, May and June, 1951, there were the usual progressive reductions in rates that occur at that time of the year.

Table C distinguishes persons who began to suffer from a serious, moderate, or mild illness during each month from those who developed an illness of a

minor or ill-defined nature. Average monthly rates during the June Quarters of 1949, 1950 and 1951 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	21–64		65 and over		21–64		65 and over	
	M	F	M	F	M	F	M	F
Apr.–June, 1949	3·2	4·0	4·0	5·5	28·6	38·3	28·0	31·2
Apr.–June, 1950	4·4	4·9	4·9	7·6	31·1	39·3	30·5	35·7
Apr.–June, 1951	4·4	5·4	6·6	9·4	32·3	39·8	32·6	34·5

There was a tendency for the rates of both the more serious and also the minor illnesses to be a little higher in the June Quarter of 1951 than in the June Quarters of the two previous years, but the increases were not sufficient to call for particular note.

Table D gives average monthly numbers of new illnesses from three selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the number of days of incapacity arising from each cause (including both new and continued illness). In this table the rates for the younger group relate to ages 16-64 in 1949 and 1950, and to ages 21-64 in 1951, so that the rates shown for 1949 and 1950 would have to be increased by a small fraction to be properly comparable with those for 1951 (see this Bulletin, October, 1951, page 241).

Following the influenza epidemic at the beginning of the year the incidence of influenza and colds rapidly declined, and fewer cases were reported during April-June, 1951, than in the corresponding quarter of the two previous years. Incapacity attributed to these causes was likewise low compared with the two previous June Quarters.

On the other hand the incidence of other respiratory diseases and of rheumatism tended to be a little higher in April-June, 1951, than in the corresponding months of 1949 and 1950.

Days of incapacity from all causes, per 100 persons interviewed, by sex and age are shown in the table below:—

	Ages 21–64		Ages 65 and over	
	Male	Female	Male	Female
1949				
January–March	122	127	200	220
April–June	86	75	151	165
July–September	82	70	89	99
October–December	98	99	225	302
1950				
January–March	114	117	196	231
April–June	89	76	143	133
July–September	79	62	118	87
October–December	99	96	182	176
1951				
January–March	167	177	295	271
April–June	86	68	126	112

In each group the average monthly incapacity rate in the second quarter of 1951 was lower than a year before and in the case of elderly persons lower still than two years before. The rates during the second quarter were less than half those recorded during the first quarter of 1951.

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 21–64						Ages 65 and over					
	M			F			M			F		
	1949	1950	1951	1949	1950	1951	1949	1950	1951	1949	1950	1951
January	35	33	28	25	25	21	20	21	15	12	12	8
February... ...	34	33	31	23	26	24	18	20	16	9	11	10
March	34	37	33	23	27	26	16	20	14	12	11	12
April	39	37	36	26	29	29	19	18	20	12	11	12
May	39	41	40	28	28	30	23	21	23	15	12	12
June	42	42	41	30	30	32	24	21	23	14	12	13
July	43	40		31	31		26	22		14	12	
August	43	42		31	31		26	25		16	11	
September ...	44	39		31	29		25	20		15	13	
October	39	36		26	25		24	22		14	11	
November ...	35	37		26	27		23	22		13	11	
December ...	36	34		27	26		27	20		14	12	

(Based on combined results of interviews in two following months.)

B.—*Monthly Illness Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 21–64				Ages 65 and over			
	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month
1949								
January ...	44·4	25·2	112	48	44·2	39·7	217	72
February ...	46·9	24·7	132	51	44·7	41·3	207	71
March ...	45·9	25·7	131	47	43·7	42·1	211	83
April	39·7	27·9	87	41	38·5	46·5	175	74
May	38·3	28·0	76	41	33·1	48·1	163	61
June	34·5	28·9	78	39	32·6	47·8	142	60
July	33·5	29·4	79	42	33·1	46·6	118	63
August ...	32·8	29·5	72	38	32·7	46·9	71	54
September ...	36·1	26·1	75	39	35·9	44·6	96	60
October ...	44·0	23·3	94	43	40·6	40·9	212	59
November ...	47·0	21·7	105	42	42·8	39·4	293	73
December ...	50·9	20·5	102	42	46·3	36·5	323	65
1950								
January ...	43·9	23·2	115	43	42·2	39·7	212	68
February ...	47·1	22·9	119	51	43·9	40·3	211	77
March ...	44·8	22·9	111	50	42·2	43·3	221	87
April	41·7	24·7	87	42	42·6	43·5	183	85
May	41·2	24·2	80	43	40·5	43·9	133	80
June	37·6	25·9	80	43	36·9	47·0	94	64
July	38·2	25·4	71	38	39·5	44·1	76	65
August ...	38·4	24·3	64	37	37·5	44·9	104	62
September ...	42·5	23·3	75	41	39·8	44·1	120	73
October ...	47·6	21·7	85	44	44·8	39·5	137	61
November ...	47·3	20·6	90	43	45·5	38·4	170	70
December ...	50·4	19·4	117	42	48·0	35·7	227	73
1951								
January ...	56·5	18·5	233	60	54·9	33·7	371	88
February ...	49·7	22·0	166	54	47·4	39·5	271	87
March ...	47·9	22·6	118	49	45·7	41·5	202	80
April	43·0	24·1	85	45	41·1	43·2	153	74
May	40·7	24·1	79	42	42·3	40·6	112	70
June	40·1	23·0	66	39	42·4	40·2	87	65
July								
August ...								
September ...								
October ...								
November ...								
December ...								

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury see Registrar General's Quarterly Returns, Table E.)

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 21–64		Ages 65 and over		Ages 21–64		Ages 65 and over	
	M	F	M	F	M	F	M	F
1949								
January ...	5·2	5·7	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	8·1	6·8	10·9	37·3	41·5	31·6	38·5
March ...	5·8	7·8	7·9	11·7	35·5	41·8	34·3	33·0
April	3·6	4·7	6·2	6·4	30·1	40·0	30·3	33·4
May	3·4	3·8	2·9	3·9	29·5	39·1	28·7	30·3
June	2·6	3·6	3·0	6·3	26·2	35·7	24·9	29·9
July	2·7	3·8	3·7	6·9	25·6	33·9	26·2	28·5
August ...	2·7	3·6	4·4	4·0	25·4	33·1	27·8	29·1
September ...	3·2	4·2	2·7	4·8	28·1	35·8	31·5	32·3
October ...	3·6	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·8	5·4	8·5	9·7	38·1	45·0	32·7	34·1
December ...	4·5	6·8	5·4	9·7	42·3	47·7	32·4	42·2
1950								
January ...	3·7	5·8	9·6	8·0	35·5	42·0	28·0	37·2
February ...	6·0	8·0	9·2	9·2	36·5	43·0	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·6	42·0	29·4	33·8
April	5·4	5·6	5·5	10·6	33·3	38·8	33·7	34·4
May	4·6	4·9	5·5	6·6	31·8	40·4	29·8	37·8
June	3·2	4·1	3·6	5·7	28·3	38·8	27·9	34·9
July	3·3	4·5	6·1	6·6	30·5	37·6	27·0	37·6
August ...	3·2	4·3	4·6	5·7	29·7	38·9	26·2	36·9
September ...	4·3	4·8	5·5	7·0	34·1	41·3	31·0	35·0
October ...	5·5	7·0	8·6	10·0	38·3	43·8	32·5	37·1
November ...	6·6	7·6	9·7	10·7	36·0	43·8	32·9	36·7
December ...	9·5	10·9	12·1	17·0	37·0	42·8	29·8	35·8
1951								
January ...	15·6	19·5	18·2	27·2	38·0	39·6	31·2	31·9
February ...	10·4	12·5	13·5	16·2	35·2	40·8	31·6	33·0
March ...	7·1	8·8	11·3	13·2	36·9	42·4	32·3	34·0
April	4·8	6·1	8·4	10·9	34·0	40·6	29·7	32·3
May	4·6	5·4	6·5	8·9	30·9	39·9	33·8	34·9
June	3·9	4·6	5·0	8·5	32·0	39·0	34·4	36·2
July								
August ...								
September ...								
October ...								
November ...								
December ...								

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. " Ill-defined " excludes all symptomatic illness which caused incapacity for Work, such cases being classed to the appropriate higher category. " Illnesses starting in each month " include new and recurrent illnesses.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 21–64*		65 and over		Ages 21–64*		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1949								
January–March ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...	19·3	19·5	13·9	13·7	15·7	22·3	36·8	31·6
1951								
January–March ...	22·9	22·5	17·2	18·0	67·3	81·4	78·3	103·2
April–June	7·6	6·5	6·2	4·6	5·6	5·3	9·4	5·9
July–September ...								
October–December ...								
	Other respiratory disease							
1949								
January–March ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...	5·6	6·6	7·4	7·1	15·5	15·5	35·1	48·6
1951								
January–March ...	6·4	6·7	8·5	7·9	25·3	30·2	83·5	73·5
April–June	4·9	4·7	6·3	6·2	14·0	6·4	25·8	20·5
July–September ...								
October–December ...								
	Rheumatism, all forms							
1949								
January–March ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	9·2	22·2
July–September ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...	6·5	9·1	9·3	13·1	4·7	5·8	6·3	16·9
1951								
January–March ...	8·2	10·5	10·4	14·5	10·9	7·3	18·6	18·0
April–June	6·3	9·1	11·3	15·0	4·8	5·2	8·5	11·0
July–September ...								
October–December ...								

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e. new, recurrent, or continued from the previous month).

* Rates for 1949 and 1950 include persons aged 16–20.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, DECEMBER, 1951

(Issued from the General Register Office, Somerset House, W.C.2)

	Dec. 1	Dec. 8	Dec. 15	Dec. 22	Dec. 29	Average weekly figures for Dec., 1950
Scarlet Fever	1,588	1,561	1,543	1,555	1,095	1,238
Whooping Cough	1,852	1,930	1,929	1,826	1,402	4,170
Diphtheria	45	30	42	54	43	46
Measles, excluding Rubella ...	2,313	2,344	2,448	2,324	2,331	14,635
Acute Pneumonia	432	462	583	683	678	673
Meningococcal Infection ...	30	29	29	30	26	32
Acute Poliomyelitis (Paralytic) ...	39	37	36	22	17	64
„ „ (Non-paralytic)	17	16	8	6	3	26
Ophthalmia Neonatorum ...	41	30	37	27	20	30
Puerperal Pyrexia and Puerperal Sepsis	218	219	226	203	131	72
Dysentery	327	537	337	422	293	949
Paratyphoid	18	14	17	17	7	5
Typhoid	2	3	1	6	3	3
Smallpox	—	—	—	—	—	1

No cases of Cholera or Plague or Typhus Fever.

VENEREAL DISEASES

Analysis of the total number of new patients attending the clinics in England and Wales during the quarter ending 30th September, 1951.*

Number of patients* attending for the first time during the three months ending 30th September, 1951, and diagnosed as follows:	M	F	M	F	M	F	Totals
(*a*) Syphilis, primary	196	27					
(*b*) Syphilis, secondary	83	85	345	186			
(*c*) Syphilis, latent in 1st year of infection	66	74					
(*d*) Syphilis, cardio-vascular† ...	80	32					
(*e*) Syphilis, of the nervous system†	151	86	631	604	1,086	963	2,049
(*f*) Syphilis, all other late or latent stages†	400	486					
(*g*) Syphilis, congenital (under 1 year)	16	34	110	173			
(*h*) Syphilis, congenital (over 1 year)	94	139					
(*i*) Gonorrhoea					4,254	901	5,155
(*j*) Chancroid					90	3	93
(*k*) Lymphogranuloma inguinale					14	1	15
(*l*) Granuloma venereum ...							
(*m*) Non-gonococceal urethritis (males only)					3,154		3,154
(*n*) Any other conditions requiring treatment					3,022	2,278	5,300
(*o*) Conditions not requiring treatment					6,799	2,871	9,670
(*p*) Conditions still remaining undiagnosed					293	214	507
					18,712	7,231	25,943

* Patients who have previously received treatment for the same condition at any Treatment Centre, or by a General Practitioner approved under Ministry of Health Circular 2226 are excluded.

† In order to avoid duplication, patients with cardio-vascular syphilis who are also suffering from syphilis of the nervous and/or other systems are recorded as suffering from cardio-vascular syphilis alone.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1

ISOLATION OF β-HAEMOLYTIC STREPTOCOCCI FROM NOSE AND THROAT SWABS BY AEROBIC AND ANAEROBIC INCUBATION

G. T. Cook, M.D., and W. H. H. Jebb, M.D.,
Public Health Laboratory, Oxford

It has for some time been the practice in this laboratory to incubate, both aerobically and anaerobically, blood agar plates spread with nose and throat swabs sent in for the detection of haemolytic streptococci.

In view of the earlier findings of Fry (1933) and Fuller and Maxted (1939) that certain strains of streptococci produced β-haemolysis on blood agar plates under anaerobic conditions only, it was thought that an attempt to compare the relative efficiency of the two methods of incubation in detecting colonies of β-haemolytic streptococci, especially those of Groups A, C and G, would be of interest.

Methods

Each swab was inoculated on to two plates of a medium consisting of 5 per cent. horse blood agar layered on nutrient agar containing digest broth. These primary plates were incubated at 37°C., one aerobically and the other anaerobically in a McIntosh and Fildes jar, and inspected the following morning. To allow for any advantage that the plate inoculated first might have, the first of the two plates was incubated aerobically during one half of the investigation and anaerobically during the other. From all cultures suspected of being β-haemolytic streptococci a single colony was picked on to a fresh blood agar plate (secondary plate) which was incubated in the same way as the primary plate, i.e. sub-cultures from aerobic plates were incubated aerobically and those from anaerobic plates anaerobically. Colonies showing β-haemolysis on either the aerobic or anaerobic secondary plates were picked into 10 ml. of 0·2 per cent. glucose broth for grouping by Lancefield's method using sera for Groups A, C and G. In addition, colonies showing β-haemolysis on the anaerobic plates were subcultured on to a blood agar plate, which was incubated aerobically. The diagnosis of β-haemolytic streptococci was, in most instances, based on the colonial and haemolytic appearances on the primary and secondary blood agar plates. In a few instances stained films of the colonies were also examined.

An attempt was made to assess the grade of growth of β-haemolytic streptococci on the primary plates, based primarily on the proportion of haemolytic streptococci present. Two grades were recorded: (1) "heavy", when the proportion of β-haemolytic streptococci was 50 per cent. or more, and (2) "light", when the proportion was less than 50 per cent. of the total growth on the plate.

Results

During the period of the investigation β-haemolytic streptococci were cultivated from 300 swabs—251 from the throat, and 49 from the nose. In half of these, *viz.* 123 throat and 27 nose swabs, the aerobic plate was inoculated first, and in the other half the anaerobic plate. No significant difference in the total number of positive isolations was observed between

these two groups, though a greater number of "heavy growths" were obtained anaerobically when the aerobic plate was inoculated first than when the anaerobic plate was inoculated first.

TABLE I

Comparison of positive isolations of β-haemolytic streptococci obtained by aerobic and anaerobic incubation of 251 *throat swabs and* 49 *nasal swabs.*

	Number of throat swabs positive			Number of nasal swabs positive			Total number of throat and nasal swabs positive by both methods
	on aerobic incubation	on anaerobic incubation	by both methods	on aerobic incubation	on anaerobic incubation	by both methods	
β-haemolytic streptococci, Group A ...	161	166	156	33	43	32	188
β-haemolytic streptococci, Group C ...	12	11	11	1	2	1	12
β-haemolytic streptococci, Group G ...	11	14	11	0	0	0	11
β-haemolytic streptococci not belonging to Groups A, C or G	53	48	43	2	3	0	43
β-haemolytic streptococci not isolated...	14	12	0	13	1	0	0
Total	251	251	221	49	49	33	254

Positive isolations on aerobic and anaerobic culture (*Table I*)

Out of 251 *throat* swabs from which β-haemolytic streptococci were isolated, 221 (88·0 per cent.) were positive by both methods of incubation. Of 166 swabs from which Group A β-haemolytic streptococci were obtained on anaerobic incubation, 156 (94·0 per cent.) were also positive on aerobic incubation ; of 161 swabs which yielded Group A β-haemolytic streptococci on aerobic incubation, 156 (96·9 per cent.) were positive on anaerobic incubation. There is no statistically significant difference between the percentages obtained by these methods. There was also no significant difference between the isolation rates on aerobic and anaerobic culture among haemolytic streptococci of Group C or G.

With *nasal* swabs, however, anaerobic incubation conferred a definite advantage. Of the 43 swabs from which Group A β-haemolytic streptococci were isolated on anaerobic incubation, only 32 (74·4 per cent.) were positive on aerobic incubation ; whereas of the 33 swabs which yielded Group A β-haemolytic streptococci on aerobic incubation, 32 (97·0 per cent.) were also positive on anaerobic incubation. The difference between these percentages is significant. In the other groups the numbers are too small for detailed consideration.

TABLE II

Comparison of grades of growth of β-haemolytic streptococci from 254 swabs (221 throat and 33 nasal) positive after both aerobic and anaerobic incubation.

	Total number of swabs positive by both methods of incubation	Number of throat and nasal swabs yielding a "heavy" growth of β-haemolytic streptococci		
		on aerobic incubation	on anaerobic incubation	by both methods of incubation
β-haemolytic streptococci, Group A	188	107	121	101
β-haemolytic streptococci, Group C	12	7	8	7
β-haemolytic streptococci, Group G	11	4	5	4
β-haemolytic streptococci not belonging to Groups A, C or G	43	12	19	12
Total	254	130	153	124

Grade of growth on aerobic and anaerobic culture (Table II)

The grade of growth obtained on aerobic and anaerobic culture has been compared for those 254 nose and throat swabs yielding β-haemolytic streptococci by both methods ; the results from nose and throat swabs showed the same pattern and they have therefore been considered together here. Of the 153 swabs which gave a heavy growth of β-haemolytic streptococci on anaerobic incubation, 124 (81·0 per cent.) gave a heavy growth on aerobic incubation, whereas of the 130 swabs which gave a heavy growth on aerobic incubation 124 (95·4 per cent.) also gave a heavy growth on anaerobic incubation. With Group A β-haemolytic streptococci aerobic incubation yielded a heavy growth in 83·5 per cent. of the cases in which anaerobic incubation gave a heavy growth, and anaerobic incubation in 94·4 per cent. of the cases in which aerobic incubation yielded a heavy growth. The difference between these two sets of percentages is significant in each case.

Conclusions

So far as throat swabs are concerned our findings suggest that anaerobic has little advantage over aerobic incubation for the isolation of β-haemolytic streptococci ; with nasal swabs, however, anaerobic incubation was of definite value and increased the number of positive isolations by approximately one-third. A heavier growth of β-haemolytic streptococci was obtained on anaerobic incubation than on aerobic in all cases, both nose and throat, in which a positive finding was obtained by both methods. This was chiefly owing to the suppression of staphylococcal and other colonies which on aerobic culture frequently obscured light growths of haemolytic streptococci. Anaerobic incubation has the additional advantage of accentuating the zones of β-haemolysis and, in doubtful cases, facilitating their distinction from α-haemolysis.

It can be seen from Table I that 6 swabs positive for Group A β-haemolytic streptococci (5 throat and 1 nasal) would have been missed if plates had been incubated anaerobically only, and that 21 (10 throat and 11 nasal) would have been missed if plates had been incubated aerobically only. In no instance did we isolate β-haemolytic streptococci on anaerobic incubation which failed to produce β-haemolysis when subsequently grown aerobically.

So far as the isolation of β-haemolytic streptococci is concerned, therefore, it seems fair to conclude that, though incubation by both methods will lead to a higher number of positives than by either alone, anaerobic incubation is to be preferred to aerobic, particularly for nasal swabs.

Summary

Nose and throat swabs submitted for detection of β-haemolytic streptococci were cultivated aerobically and anaerobically on blood agar plates. The results indicate that anaerobic is to be preferred to aerobic incubation, particularly for nasal swabs, but that some positive swabs would have been missed if one or other method had been omitted.

We are indebted to Dr. Ian Sutherland, of The Institute of Social Medicine, Oxford, for statistical advice.

References

Fry, R. M. (1933) *J. Path. Bact.*, **37**, 337.

Fuller, A. T. and Maxted, W. R. (1939) *J. Path. Bact.*, **49**, 83.

A NEW SALMONELLA TYPE ISOLATED FROM A BABY : SALM. CLERKENWELL

P. Story, M.D., St. Bartholomew's Hospital, London, S. Hilda Douglas, M.Sc. and Joan Taylor, B.Sc., M.B., B.S., D.P.H., Salmonella Reference Laboratory, Colindale, London.

A female baby aged three months was seen in the Children's Out-patient Department at St. Bartholomew's Hospital. Her illness started one morning with abdominal colic which " doubled her up ", and during the day she passed three green stools containing blood and mucus. There was no vomiting or loss of appetite. She was a bottle-fed baby and had thriven steadily since birth.

The child appeared healthy and well nourished and a complete physical examination revealed no abnormality except shallow ulceration of the perianal skin. She was given only boiled water for twenty-four hours followed by increasing strengths of cows' milk, and recovery was rapid ; by the fifth day the stools looked normal. Rectal swabs and faeces were cultured and an organism of the salmonella group was isolated on four occasions, the first being on the 2nd and the last on the 54th day after onset of disease. Twenty days later we failed to grow the organism from a rectal swab. Serum taken on the 15th day contained agglutinins: O – 1/50 ; H (z) – 1/50 ; H (l, w) – nil 1/25.

The child's home was visited in an attempt to trace the source of infection. As far as we could ascertain there was no animal contact of any kind except the family cat ; and culture of a rectal swab taken from this animal yielded no growth of *Salmonella.* Bacteriological examinations of human contacts and of food were negative except for the baby's mother, from whose stool we isolated a *Salmonella* identical with that of the child. Two weeks later culture of the mother's faeces failed to reveal this organism, and no agglutinins were detectable in her serum when tested for at a dilution of 1/25 with O, H (l, w), and H (z). The mother had suffered a mild attack of enteritis about three months previously, but none of the other family contacts gave a history of recent diarrhoea.

Bacteriology

Three strains isolated on two separate dates from the baby and three strains isolated from one specimen of the mother's faeces were studied and found to be identical both in antigenic structure and biochemical behaviour. The organism, a Gram-negative motile bacillus, produced a typical salmonella colony on MacConkey agar. Glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose, trehalose and inositol were fermented in 24 hr. with the production of acid and gas. Lactose, sucrose, salicin, adonitol, inulin and raffinose were not fermented in 25 days. Koser's citrate and Jordan's tartrate utilization tests were positive, and H_2S was produced ; indole was not formed ; urease was not produced (Christensen's medium) ; and gelatin was not liquefied in 22 days. The Voges-Proskauer reaction was negative and the methyl-red reaction positive.

Serological examination showed that the organisms were agglutinated by *Salm. anatum* O serum to titre. A suspension of one strain removed all O agglutinins from *Salm. anatum* serum, and similarly *Salm. anatum* removed all O agglutinins from a serum made with the new type. Therefore the somatic structure is represented by the symbols iii.x.

The organism was diphasic and both H phases could be isolated from primary cultures. Phase I was agglutinated to titre by *Salm. london* H (l, v) serum. When tested with monospecific sera for factors v and w, it was agglutinated by w. *Salm. dar-es-salaam* H (l, w) serum agglutinated the new type to titre and the new type absorbed all homologous l, w agglutinins from the serum. A serum made from the new type agglutinated *Salm. dar-es-salaam* H (l, w) to titre and a suspension of the latter removed all homologous H (l, w) agglutinins from this serum. Therefore the antigenic structure of Phase I is represented by the symbols l, w. Phase II was agglutinated to titre by *Salm. poona* H (z) and by *Salm. worthington* H (z) sera. Absorption of *Salm. poona* serum with the new type reduced the homologous titre from 1:25,000 to 1:1,600; this suggests that there is some minor antigenic difference between the H (z) of *Salm. poona* and the new type. Absorption of *Salm. worthington* H (z) serum with the new type removed all homologous agglutinins; similarly *Salm. worthington* H (z) removed all H (z) agglutinins from a serum made with the new type.

Summary

A new salmonella type has been isolated from the faeces of a baby suffering from diarrhoea, and from the faeces of the mother.

The antigenic structure of the organism is iii.x: l, w—z, and the suggested name is *Salm. clerkenwell.*

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health,
Savile Row (Room 207),
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The December Bulletin was issued on 21st December.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(14219) Wt. 8055—6219 3200 1/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

FEBRUARY **1952** **Vol. 11**

CONTENTS

Section I

Section II

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1

DIPHTHERIA IMMUNISATION INQUIRY

P. G. Gray, B.Sc., and Ann Cartwright, B.Sc.
Government Social Survey

Purpose of the Inquiry

Immunisations of children under five years numbered only 433,000 in 1950, compared with 574,000 in 1949—a drop of 141,000. Only about 34,000 of this 141,000 could be accounted for by the reduced birth-rate and consequently fewer children needing immunisation. The Ministry therefore asked the Social Survey to carry out an inquiry to enable the Ministry "to assess more exactly the new problems of diphtheria immunisation publicity and to make whatever changes or adjustments may seem necessary in the publicity approach". One problem in mind was the possible deterrent effect of the observed occasional association between immunisation and poliomyelitis not only by its causing immunisation to be suspended during periods of high prevalence in many areas, but also by affecting the attitude of the mothers. Immunisation was suspended in about one-third of all areas in 1950 for periods varying from one to five summer (or near summer) months. This, we calculate, could have accounted for a fall of 45,000 in the whole country, which left us with a remainder of 62,000 unaccounted for.

Method of Inquiry

A two-fold approach was made. The mothers of a sample of children aged between six months and the fifth birthday were interviewed, and a postal questionnaire was addressed to the medical officers of health in the districts in which the mothers were interviewed. The children were selected from the Ministry of Food records in 58 administrative districts in England and Wales. Less than one per cent. of the mothers refused to co-operate. The 1,254 mothers who did were visited in their homes by trained interviewers, who introduced the survey to them as a general one relating to the health of young children, diphtheria not being mentioned at first, in order to avoid influencing the answers. The first question was a factual one about the illnesses the child had had, and after that there were three designed to find out the mother's attitude towards and fear of diphtheria and poliomyelitis in relation to other illnesses. There followed some questions to discover how much the mother knew about the ways in which children get various illnesses, and the means of prevention. Only after this was the interview focused on diphtheria and immunisation.

Findings

I. The Age of Immunisation

Age at time of interview (May 1951)	Percentage of children with completed immunisations (2 or more injections)	Percentage of children with 1 or more injection	Number in Sample
6 months—8 months	—	6%	52
9 months—11 months	28%	47%	60
1 year—1 year 5 months	56%	68%	133
1 year 6 months—1 year 11 months	65%	70%	143
2 years—2 years 11 months ...	76%	80%	275
3 years—3 years 11 months ...	79%	80%	281
4 years—4 years 11 months ...	82%	84%	310
6 months—4 years 11 months ...	69%	74%	1,254

Footnote. For further details, particularly of the sampling method and the questions asked, see "Diphtheria Immunisation in 1951", Gray, P. G. and Cartwright, Ann. Social Survey Report 55, 176, London, 1951. The Social Survey.

In May, 1951, 74 per cent. of the children aged* between six months and the fifth birthday had been, or were being immunised. The proportion with completed immunisations was 5 per cent. lower, at 69 per cent. Of those aged three to five, four out of five had been immunised. Only 47 per cent. of children aged nine months to under a year had received at least their first injection. Furthermore only 68 per cent. of those between 12 and 18 months had had one or more injection. It is clear therefore that the target of 75 per cent. immunised before their first birthday is not being attained; in fact, only for those aged two or over is this target reached for completed immunisations. Further analysis showed that the best that has been achieved was when 55 per cent. of babies received their first injection before their first birthday, this was for the age group two years to two years 11 months in May, 1951.

Where the Child was Immunised

The proportion of immunisations carried out at clinics has fallen during the last five years.

II. Where the Child was immunised

Where the immunisation was done	Percentage of all immunised Children	Year of Immunisation			
		1947	1948	1949	1950–April 1951
	per cent.	per cent.	per cent.	per cent.	per cent.
Clinic	61	69	66	62	49
Family Doctor					
At surgery	28	18	22	28	39
At child's home	7 35	10 28	6 28	7 35	8 47
Other places	4	3	6	3	4
Number of children in SAMPLE	928	157	249	242	280
Proportion of immunisations for which parents paid ...	5%	12%	8%	3%	1%

The classification by year of immunisation is inexact, depending upon the mother remembering the date of immunisation; moreover of the children immunised in 1947 not all were represented in our sample.

Of all the children aged six months to under five, who had been immunised, 61 per cent. had been done at the clinic, 28 per cent. by the family doctor at his surgery, 7 per cent. by him at the child's home and 4 per cent. at "other places", these including mobile vans, day nurseries or schools and a few at different places for successive injections.

The proportion immunised at the clinic has fallen from just over two-thirds in 1947 to about one half in 1951, while that done by the family doctor at his surgery has correspondingly increased; the proportion done by him at the child's home has remained fairly steady.

The proportion for which the parents paid has declined from 16 per cent. (Box)† in 1945 to 1 per cent. in 1951. Whereas one-tenth of those immunised at the doctor's surgery paid for it, a third of those immunised by him in their own homes did so.

* For the proportions on 31st Dec., 1951, of children of various ages immunised in each county and class of area, see the Registrar General's Statistical Review, 1949, Vol. I, Text, Appendix A, 283.

† Box, K., Diphtheria Immunisation. Social Survey report N.S.69, London, 1945, The Social Survey.

Site of Injection

Table 3 shows the different sites or combinations of sites of injection and the resulting discomfort, if any, reported by the child's mother.

III. Sites of Injections and Discomfort

Site of Injection	Percentage of children injected at site(s)	Percentages of children having trouble of different kinds			
		Soreness	Sickness	Mild Disorder	No trouble
	per cent.				
Right arm	12	11%	3%	1%	85%
Left arm	40				
Both arms	15				
Right leg	3	31%	6%	4%	59%
Left leg	4				
Both legs	2				
Buttock	11	20%	2%	3%	75%
Other combinations	3	20%	3%	4%	73%
Uncertain	10				
All immunised children ...	928 (100)	15%	3%	2%	80%

Two-thirds of the children had been injected in the arms, the left arm (40 per cent.) being clearly the most popular site. The relative popularity of different sites has little changed, though the buttock (11 per cent.) appears to be gaining slightly in favour. Injection in the arm caused the least discomfort. Whereas soreness of the child's arm was reported by 11 per cent. of the mothers, whose children had been injected in the arm, soreness of the leg was reported by 31 per cent. Only 3 per cent. of mothers considered the place of injection to have been very troublesome.

The increasing use of the buttock as a site for injection is due to the increased frequency with which combined whooping cough and diphtheria immunisations have been made; 29 per cent. of all immunisations in this sample. The proportion of mothers saying their children experienced trouble from this type of immunisation was 37 per cent. as against 16 per cent. for those immunised against diphtheria only. Although the injections for these combined immunisations tended to be made more frequently in the buttock or legs, it could be shown that, independently of each other, combined immunisation and the use of legs or buttock led to greater trouble for the child.

Reasons Given for Not having Children Immunised

In this sample of children 26 per cent. had not received even a single injection. Their mothers were asked why their children had not been immunised, and their reasons are summarised and compared with those given in the 1945 inquiry in Table 4.

IV. Mothers' Reasons for Not having Children Immunised

Percentages of children in each age group

Reasons given for not having children immunised	6 mths. to under 5 (1951)	Age at time of Interview						1945 Inquiry (Children aged 1 to under 5)
		6 mths. —11 mths.	1 yr. —1 yr. 5 mths.	1 yr. 6 mths. —1 yr. 11 mths.	2 yrs. —2 yrs. 11 mths.	3 yrs. —3 3 yrs. 11 mths.	4 yrs. —4 yrs. 11 mths.	
Too young	4	36	1	1	—	2	1	6
Child's illness	5	14	9	6	6	4	2	3
Father does not agree ...	3	4	2	4	3	2	3	3
Does not believe in it ...	2	1	2	1	1	2	3	4
Never heard of it ...	—	1	—	—	—	1	—	1
Difficult to get there ...	4	4	8	6	3	5	2	
Not bothered	3 } 11	3	3	4	3	2	3	} 20
Others	4	8	5	6	3	2	2	
Poliomyelitis	1	1	2	2	1	—	—	—
Percentage of immunised children in each age group	74	28	68	70	80	80	84	63
Number in SAMPLE ...	1,254	112	133	143	275	281	310	877

The reason "too young" was given less frequently for those over one year old than it was in 1945, and, when asked a further question, all mothers, who gave "too young" as a reason, said they intended to have their children immunised.

The proportion of cases where the father was said to disagree with immunisation varied but little in the age groups and remained the same as in 1945. Where mothers blamed father, they were asked why father objected and in a quarter of cases his experience of inoculation in the Forces was quoted or, in a further quarter, the similar experience of some other person.

The proportion of mothers who said they did not believe in immunisation was rather lower than in the 1945 inquiry. The proportion who had never heard of it had also declined, from 13 cases in 1945 to only 3 in 1951.

The four reasons "Father does not agree", "Not bothered", "Does not believe in it", and "Never heard of it" do not vary appreciably with age of the child, and together form the unsatisfactory excuses mentioned in the next section.

The last group shown in Table IV consists of those whose mothers mentioned poliomyelitis, infantile paralysis or polio in giving their reason. This group appeared of course only in the present inquiry, and in the younger age group. Although small, less than 1 per cent., this group is of interest and will be discussed in more detail later.

The Effect of Family Background on Immunisation

The family background not only affected the proportion of children who had been immunised, but also the age at which they had been immunised and the reasons given for not having some children immunised.

Space does not permit the detailed analysis made to be given here but, as would be expected, the groups of mothers least co-operative in immunisation are the less well educated, those from poorer households and those with several children. These groups have small proportions of immunised children, and even smaller of these immunised at the right age. They also give a higher proportion of unsatisfactory excuses for not having their children immunised. (A smaller proportion of their children are vaccinated against smallpox.)

The highest percentage immunised achieved by any group was 85 per cent. for the group of children with the better educated mothers. This group approached the maximum possible proportion of 93 per cent., which could be attained for the age group 6 months to under 5 only if injections started at 8 months and the proportion injected increased linearly to 100 per cent. by the first birthday. At the other extreme are the children from families with four or more children. Here only 55 per cent. are immunised, a figure which compares well with that of 50 per cent. given by Rowntree for children of fourth or higher birth orders.

Although the three characteristics, education, income, and number of children in the family are not entirely independent, each characteristic had its own effect on the proportion immunised.

Families tended to be immunised as a whole or not at all. There were in the sample 263 children who had an elder brother or sister under 5. Where the elder child had been immunised the sample child was already immunised in 70 per cent. of the cases, while a further 20 per cent. had yet to reach the age at which the older child was done. On the other hand, where the elder child had not been immunised, the child in the sample had only been immunised in 2 per cent. of cases.

The occasional association between immunisation and poliomyelitis

Of the 9 instances where poliomyelitis was referred to in the reason given for not having had the child immunised, only in 3 did the mother appear to have been frightened by the knowledge. In the other 6 cases poliomyelitis was merely mentioned as the reason for postponing immunisation.

In all these 9 cases the mothers, in reply to a further question, said they intended to have the children immunised. Whether they will, is, of course, open to question, but they do not seem very strongly opposed to it. Apart from these 9, we tried by two indirect questions to discover how many mothers were aware of the occasional association between inoculation and poliomyelitis. To avoid alarming any mother no direct question was asked.

In all, 56 mothers (4 per cent.) showed that they were aware of the association. Of the 56 children concerned, 41 (73 per cent.) had been immunised. If we consider only the 32 children born since mid-1949, i.e. those whose mothers were most likely to be affected by this knowledge, 21 (66 per cent.) had been immunised.

We conclude that the fear of contracting poliomyelitis after immunisation *does not* at present seem a factor of importance in the immunisation campaign. The majority of the medical officers of health appear to agree with this conclusion. Incidentally, no case of poliomyelitis following immunisation was reported among the sample group.

Suspension during seasonal prevalence of poliomyelitis

We have already given our estimate of the proportion of the fall (45,000) which could be attributed to this. Suspension may also account to some extent for the decline in the proportion of immunisations carried out by clinics, which appear to have considerable influence with mothers.

Other factors in the decline

We would emphasise the possibility that fear of diphtheria is declining, and that the very success of the immunisation campaign in almost eliminating diphtheria, has led to some apathy on the part of mothers, who may not realise that this is conditional upon the maintenance of an adequate level of immunisation. The cessation of the publicity campaign during the summer and autumn of 1950 may also have contributed to the decline in the fear of diphtheria. In addition, the proportion of mothers saying that diphtheria is the most dangerous illness declines with the increasing number of children in the family, while the proportion most fearing whooping cough increases slightly with the size of the family; this suggests that mothers of large families fear whooping cough more because they have had experience of it with their elder children, while they know that diphtheria had not attacked their elder children.

Means of Persuasion

Both the evidence of this survey and the opinions expressed to us by medical officers of health suggest that personal contact with and persuasion of the mother at the clinic or by the health visitor or the family doctor was of the first importance. Four per cent. of mothers could not be reached at any stage through the clinic or the health visitor.

Acknowledgments

We wish to thank all the mothers interviewed and also the medical officers of health for their co-operation in this inquiry.

FOOT HEALTH

Arthur F. Alford, M.B., Ch.B., Ministry of Education, and
Mary G. Gorrie, M.D., D.P.H., Ministry of Health.

In this Bulletin, November, 1946, Dr. J. L. Burn pointed out the need for the education of the public in foot health, for preventive measures and for chiropody. He defined chiropody, gave the costs of a clinic and analysed the conditions treated. He emphasised the need for some evening sessions.

It now seems opportune to review the subject. B. C. Bradley, K. L. C. Freeborn and J. C. R. Clapham for the British Boot, Shoe and Allied Trades Research Association (SATRA) made a survey(1) of 900 school children in 1948. They found that hallux valgus was frequent at all ages, being found in 10 per cent. of two year olds and in 88 per cent. of girls and 67 per cent. of boys over 16; 60 per cent. of boys below 15 years and 80 per cent. of all ages were wearing shoes too short for their feet.

In the Auxiliary Territorial Service during the war, observations(2) were made on the feet of 1,625 women, including 812 recruits, 560 with varying periods of service and 253 auxiliaries under treatment. Ninety-six per cent. were under 21 years, 3 per cent. between 21 and 26 and 1 per cent. over 26.

Excluding those under treatment the incidence was as follows:—

	Per cent.
Dropped long arch	25
Clawed toes	12
Dropped transverse arch and hallux valgus each ...	7
Cavoid foot	3
Overlapping toes	1–5

The foot defects were as prevalent among recruits as among the older auxiliaries, from which it seemed that in most cases the disabilities had begun in early life and that the feet had been functioning at a mechanical disadvantage.

In a sample of 439 old people,(3) Dr. J. H. Sheldon found that 26·3 per cent. of men and 46·9 per cent. of women between 65 years and 70 years were suffering from disability of the feet. He commented on the fact that life "would have been made more bearable for them were it possible for them to visit a chiropodist regularly. . . . Even more desirable however is an expert examination of the feet of a random sample of old people, which alone would provide the detailed information which is very necessary."

These figures are by no means exhaustive but they are typical and they illustrate the important point that the findings were made in the examination of ordinary *healthy* people and not among those who had presented themselves for foot treatment. Another important point is that more and more our attention is directed to early life for causes and prevention.

The Care of Feet in the Health Services

Central Government Departments

Under the Health Act chiropody is recognised as an adjunct to hospital and specialist treatment, and in collaboration with the Central Council for Health Education, the Ministry of Health has recently sponsored the film "Your Children Walking" which can be borrowed free of charge from the Central Film Library.

The Ministry of Education approves the employment of chiropodists in the school health services. Thirteen local education authorities make use of these experts.

Local Authorities

Their activities include routine foot inspections ; chiropodial treatment ; surveys such as have produced the above figures ; and certain measures such as the Ealing scheme designed to help parents to set aside the cost of suitable shoes.

In maternity and child welfare departments, the clinic talks for expectant mothers usually include a talk on feet and posture and the feet of the under fives are examined at child welfare clinics and in the homes. In any adequate school health service, the examining medical officer inspects the child in every respect, and for this purpose it is obvious that the child should be examined with *bare* feet.

These inspections serve as a screen, dividing minor ailments and abnormalities which are dealt with by officers of the local authority, from major defects referred either directly or through the patient's own doctor to a hospital orthopaedic clinic.

In the research field the surveys which are known to have been made deal mainly with the foot defects of the school child in relation to his shoes, and at least one has been concerned with the normal growth of children's feet. But the picture is by no means perfect and local authority officers will detect a number of obvious gaps.

First, up to date little attempt has been made to *teach prevention.*

Secondly, the number of medical auxiliaries employed is still small, and even curative treatment can only have touched the fringe of the problem. Although the medical officer must always be the responsible officer, the part which the chiropodist can play in the school health service is not fully realised.

Thirdly, routine examinations are not always so effective as they might be. The limits of normality are very wide and therefore many medical officers have difficulty in deciding which cases to refer to an orthopaedic consultant, and sometimes the specialists say that the wrong cases have been sent to their clinics. The shoe salesman is sometimes confronted with an instruction which is not easily understood. In their busy rounds health visitors may need to know more about the important points for which they must look.

Even where good advice has been given on hygiene of the feet and in regard to socks or stockings, a medical officer may feel obliged to gloss over or to withhold comment on " border line " shoes because of the cost of a new pair which is a very frequent and understandable difficulty.

It would be unfair to lay all the blame on the shoe trade for producing the wrong type of shoes. That menace to infancy, the ankle strap shoe with the rigid sole, still appears but in smaller numbers ; shoes, for children particularly, and for girls and women—increasingly—become suitable as well as attractive. Although there is still a great deal to be done, much has already been achieved by last and shoe manufacturers and by retailers to improve the types and the quality of shoes against the demands of fashion.

Though both in the trade and in the medical and auxiliary profession, the ways to improvement have already been opened, their efforts should be intensified. Educational programmes such as those outlined by Dr. Hamilton Hogben at the recent Food Health Educational Bureau Conference in October, 1951, might well be increased and be preceded by talks to medical

officers and health visitors. Usually there will be one medical officer on the local authority staff who is willing to take part in lecturing or in leading a discussion, and where there is a chiropodist, he should make a valuable contribution.

The medical officer of health might consider inviting an orthopaedic surgeon to lecture to his medical staff.

Any such lecture or discussion might well be attended jointly by medical officers, health visitors and school nurses. Medical officers and health visitors should be recommended to study the F.H.E.B. pamphlets,(4) particularly " The Foot and the Shoe " by T. T. Stamm, F.R.C.S. (1947), and " Foot Inspection in Childhood and Adolescence " (1949).

The vital importance of care of the feet, including the choice of footwear, should be emphasised to parents and older children. This may encourage them to endure the high cost of shoes, but it is to be hoped that some means can be devised which will help in reducing this very serious drain on the family budget, which is annoying when, as often happens, the child out-grows his shoes before they are worn out. Nor in giving advice should the size and care of socks be forgotten.

The film " Your Children Walking " shows the inter-dependence of feet, posture and health, and it illustrates foot toilet and the selection of footwear. It should be a help to officers who are trying to teach prevention: it was planned to show at welfare clinics and deals primarily with the mother's care for her young child ; but it may well be more widely used to encourage older people to choose the right type of footwear and to realise the importance of timely repair and replacement. The importance of active muscle control is emphasised in the film, a good point when directing parents' attention to posture as well as choice of footwear.

There is much to be done in the clinical fields from correlated research among children into growth rate, the influence of heredity, and the effects of early abnormality upon defects of later life, up to old age, where there is wide scope for investigation and care which would be of economic benefit to the community, as well as bringing comfort and prolonged mobility to the individual.

Education of the public should include our own education and should promote an interchange of information and ideas between the different bodies who are interested in feet and footwear, and, in time, may lead to a change in Fashion, that wayward lady who is justly blamed for so many defects.

The *first* duty in foot care, as in all medicine, is *to prevent*.

References

(1) Report of the British Boot Shoe and Allied Trades Research Association, Research Report, No. R.R. 107.

(2) Personal Communication from Dr. (Major R.A.M.C.) Doris Baker—unpublished survey: " Foot Defects in the A.T.S."

(3) The Social Medicine of Old Age. J. H. Sheldon, M.D.(London), F.R.C.P.(London), Report of an enquiry in Wolverhampton for the Nuffield Foundation. Oxford University Press 19 & 8.

(4) Foot Education Bureau, 121, Ebury Street London, S.W.1, (1/- each, post free).

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

JANUARY, 1952

(Issued from the General Register Office, Somerset House, W.C.2)

	January 5th	January 12th	January 19th	January 26th	Average weekly figures for January 1951
Scarlet Fever	1,104	1,055	1,211	1,293	937
Whooping Cough	2,155	2,394	2,349	2,573	4,959
Diphtheria	45	33	22	27	40
Measles, excluding Rubella ...	3,491	2,840	2,828	2,837	18,072
Acute Pneumonia	976	1,058	869	836	2,749
Meningococcal Infection ...	40	51	35	37	54
Acute Poliomyelitis (Paralytic)...	18	33	25	18	28
,, ,, (Non-paralytic)	7	3	10	9	9
Ophthalmia Neonatorum ...	24	34	36	30	22
Puerperal Pyrexia and Puerperal Sepsis	197	247	232	207	100
Dysentery	295	315	434	547	741
Paratyphoid	6	3	6	10	4
Typhoid	2	3	4	2	3
Smallpox	—	—	—	—	7

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Harrogate Laboratory : telephone number

The laboratory now has an independent telephone line. The number is : Harrogate 84077.

AN OUTBREAK OF WATER-BORNE GASTRO-ENTERITIS AND SONNE DYSENTERY

A. I. Ross, M.D., D.P.H., Deputy Medical Officer of Health, City of Leicester, and E. H. Gillespie, M.B., Ch.B., Director, Public Health Laboratory, Leicester.

As Glover (1947, 1949) has pointed out, many hundreds of cases of bacillary dysentery occur in this country yearly. Outbreaks are usually due to infection from food, and are common in institutions. Hobbs and Allison (1942) have stressed the importance, in the spread of the disease, of " missed " cases, the symptomless carrier (often an adult) and the premature release from isolation of convalescent carriers. In this country in recent years, as far as we have been able to ascertain, only one outbreak has been conclusively proved to have been due to water-borne infection. For that reason it has been thought desirable to record an outbreak of water-borne gastro-enteritis which included cases of Sonne dysentery, occurring in Leicester in December, 1950.

The Outbreak

Onset

On the morning of 20th December, 1950, the safety officer of a large factory, after notifying the works doctor, telephoned the Health Department to say that 18 of the 1,250 employees were suffering from vomiting and diarrhoea. The factory was immediately visited and a description of the outbreak obtained. It was stated that the water was under suspicion as some of the employees had noticed that it smelled badly. Two samples of water from taps in the canteen and works surgery were inspected, and found to be very slightly cloudy, but a smell was not detected. Samples of the water in the canteen were taken for bacteriological and chemical analysis.

Clinical details

By the time the factory had been visited, more cases of sickness had been reported among the employees in various parts of the works, of whom twenty were interviewed.

The earliest symptoms began at 7.0 p.m. on the 19th, and the latest started just before 11 a.m. on the 20th, *i.e.*, the day the outbreak was reported. The symptoms were vomiting with diarrhoea and colicky abdominal pains. Two or three of the patients suffered from vomiting only and one or two from diarrhoea only ; none had vomiting or diarrhoea more than seven or eight times. There were few systemic symptoms apart from slight giddiness and headache.

Action taken

A full history of everything that had been eaten and drunk in the works by all the patients within the previous 48 hours showed that all, with one exception, had drunk tap water. There appeared to be no other common factor. Many had had nothing to eat or drink from the factory canteen.

On inspection, the canteen was found to be scrupulously clean and well run. There was no food left over from the previous day except one half empty packet of dried egg. Only one of the canteen staff had been affected. Specimens of vomit and faeces for bacteriological examination were requested from as many of the affected persons as possible, and three stool specimens from all canteen staff.

The works manager and the works engineer stated that the factory obtained its water from two sources: (1) Leicester city water was piped direct at 25–30 lb. pressure to various parts of the factory and to four 80-gallon storage tanks over the canteen; (2) River water was distributed at 240 lb. pressure and used untreated for industrial cooling and hydraulic presses. The engineer stated that there was no possibility of the two supplies becoming mixed as no work had been undertaken lately which would cause this (but see later). We already knew that the town mains in the neighbourhood were in good condition, that they had not recently been interfered with, and that all the samples of Leicester water from this district tested as a routine had been satisfactory.

Cause of the outbreak

The following morning (21st) the result of the bacteriological examination of the water sample from the canteen gave a presumptive coliform count of 180 + per 100 ml., indicating gross contamination. The works manager was immediately requested to stop the use of piped water for drinking and dish-washing and to provide boiled water for these purposes. Arrangements were made to inspect the water supply with officers of the Leicester Water Department. Particulars had been obtained of 40 more patients who had developed vomiting and diarrhoea during the night and morning. It was not known how many had stayed away from work as a result of these symptoms. Along with officers of the Corporation water department, further samples of the water from the canteen were taken and the storage tanks inspected. Two of these had a slight scum on top and were connected to the other two in such a way as to prevent adequate circulation of water.

The town water supply in the works was now perfectly clear. The works manager had already taken action, as requested, to boil water, and explanatory notices were prominently displayed throughout the works. At this stage we were little further towards finding the source of infection.

By the afternoon of the same day the City Analyst had completed the chemical analysis of the water taken from the canteen the day before. His results showed the water to be grossly contaminated and inconsistent with its being derived from any of the sources of Leicester's drinking supply (Table 1).

TABLE 1. *Chemical Analysis of the Sample of Canteen Water*

	Analysis of sample of water taken from tap in works canteen on 20.12.50	Typical results of River Soar Water	Usual figures for Leicester Water
Appearance	Opalescent	Brownish	Clear and bright
Hazen No.	About 50	60	5 to 14
pH	7·7	7·5	7·1 to 8·8
Chloride	27·0 p.p.m.	44·0 p.p.m.	10 to 21 p.p.m.
Free Ammonia	0·30 ,,	1·0 ,,	0·006 to 0·01 ,,
Hardness	275 ,,	340 ,,	40 to 180 ,,
Total Solids	457 ,,	565 ,,	80 to 300 ,,
Nitrite	Present	Present	Absent

Lead and arsenic were not detectable; zinc was present in less than 0·1 part per million, copper amounted to 0·04 part per million, and iron 0·4 part per million.

On receiving information that river water was also used in the works for certain purposes, the City Analyst had taken a sample from the river near the works, and found it to be so similar in appearance to the sample of canteen water and to give such close figures to it on chemical analysis, as to prove fairly conclusively that the sample from the canteen was, in fact, river water and not City supply.

A further visit was made to the works and the works manager and engineer informed of the results of the chemical analysis. The engineer then stated that the river water, because of the dirt it contained, had been found unsuitable for the particular manufacturing process undertaken in Room 53 of the factory, and for that reason on Sunday, 17th December, a connecting pipe had been placed between a river water supply pipe and a town supply pipe. A valve had been fitted in the connecting pipe to prevent any communication between the two supplies (Fig. 1). The engineer had intended later to disconnect the river water pipe from the river supply and run town water into the river water pipes in this room. Unfortunately, unknown to him, the workman who had fitted the connecting pipe had left the valve open, so that from 6 a.m. on Monday, the 18th, when the pumps were started, until 10 a.m. on Wednesday, the 20th, when the cross-connection was removed, river water at 240 lb. pressure had been forced into the town supply, which was at 25–30 lb. pressure.

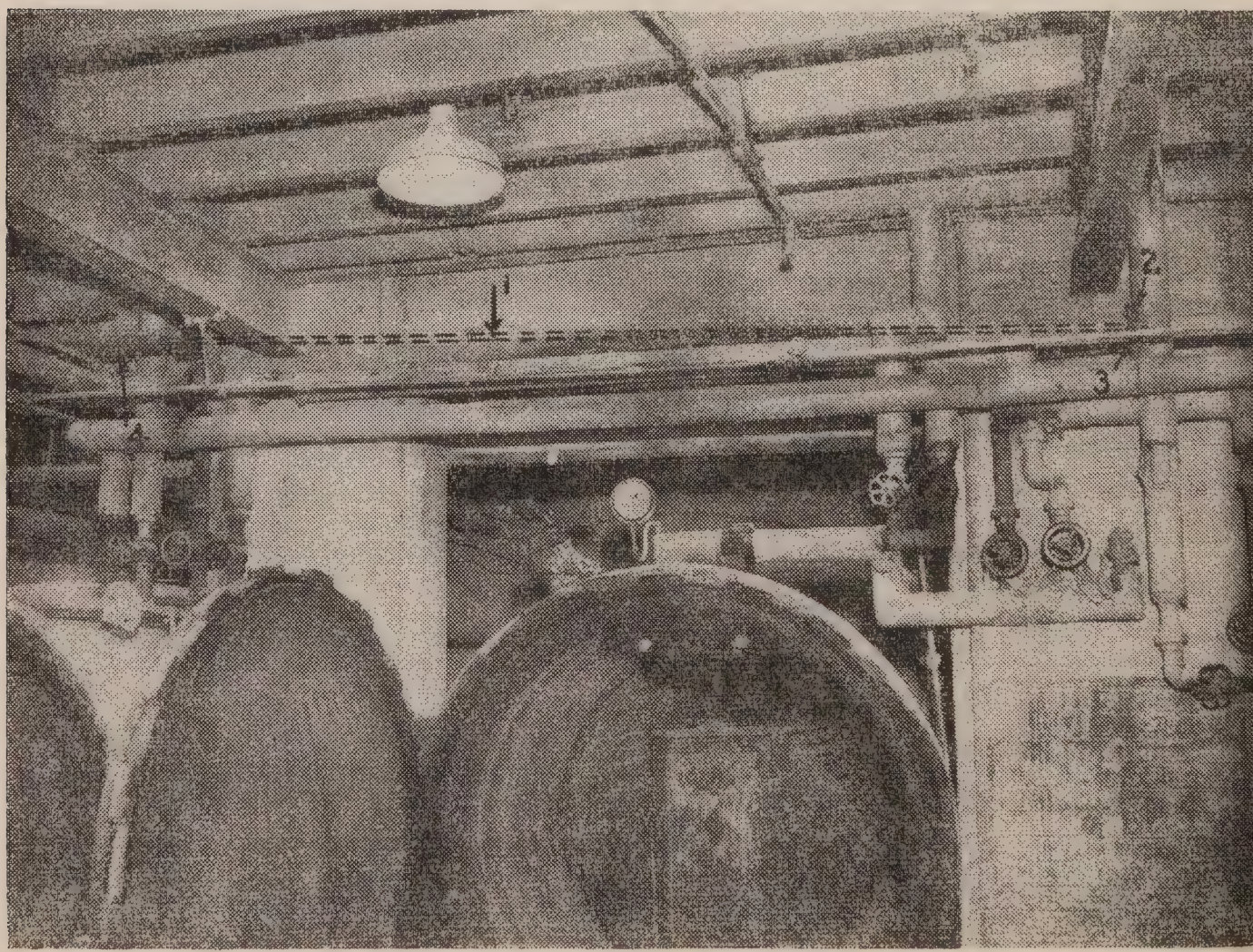


FIG 1. Photograph showing line of intercommunication between the two pipes.

1. Line of intercommunication
2. Valve
3. River water main
4. Town main

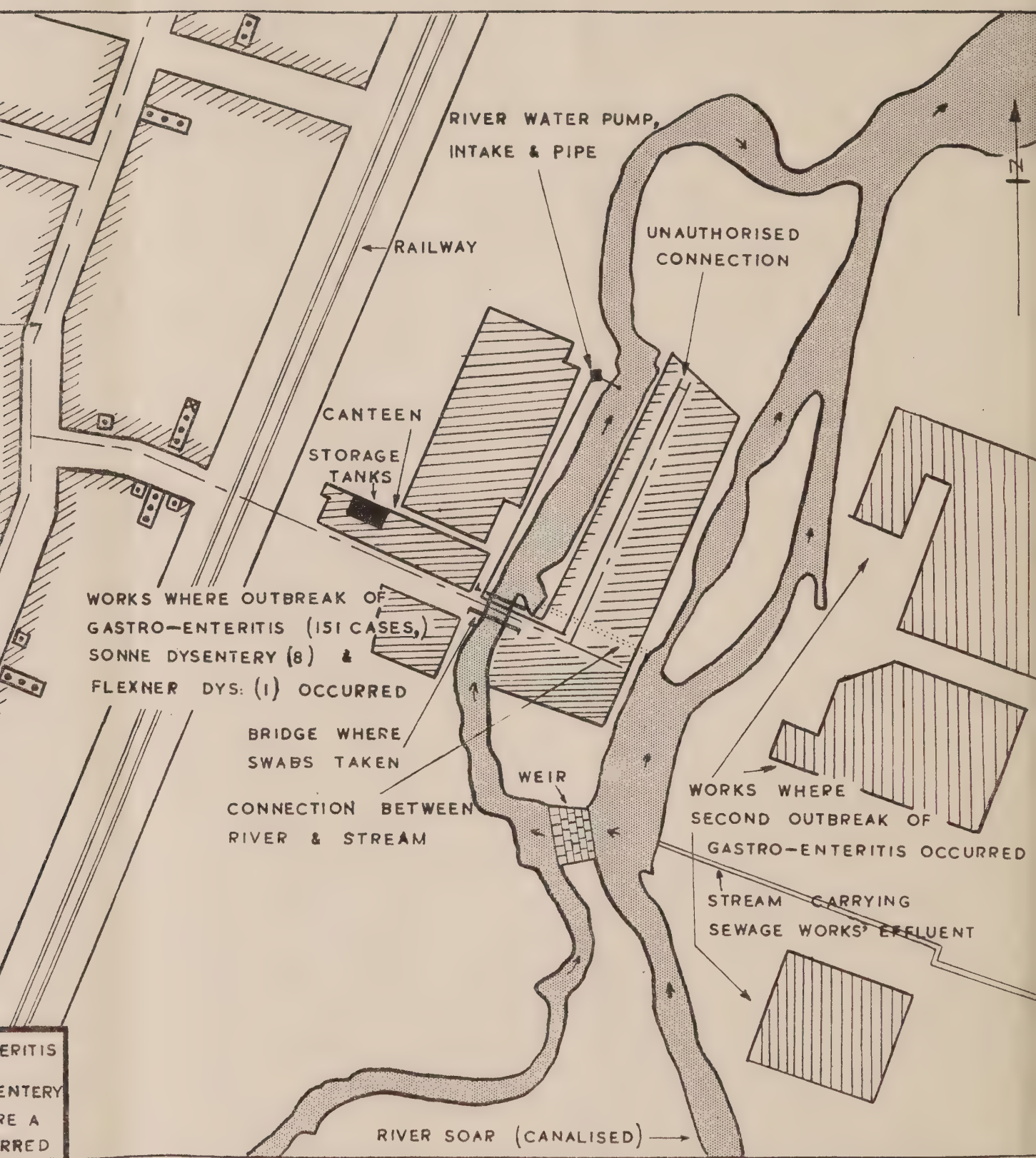


FIG 2. Plan of the factory.

[*To face page 39*

As shown in the plan of the factory and its surroundings where the water distribution pipes are indicated, it appeared probable that because of the increased pressure the river water had been forced some distance back into the town mains, contaminating the supply to nearby houses. Accordingly it was arranged with the waterworks engineer to flush the mains and also the pipes in the works and to superchlorinate the water. All nearby householders were requested to boil their water until further notice.

An account of the outbreak was sent to the general practitioners in the city and they were warned about the possibility of enteric fever occurring later.

Length of time town mains took to clear

The cross-connection was removed at 10 a.m. on the 20th. As stated above, samples taken in the middle of the morning from the canteen tap were highly contaminated. A sample taken from the canteen on the 21st at 11.45 showed 90 coliform organisms per 100 ml. After flushing and chlorination on the 21st, samples were obtained on the 22nd from three houses near the factory. Two were satisfactory and one showed 8 coliform bacilli per 100 ml. All samples on the 27th were satisfactory.

Further clinical details

Later, when as many as possible of those employees who had been ill were interviewed, and house-to-house visits were paid near the works, we found that 160 employees and 28 people living nearby and not working in the factory had been ill. Three specimens of stools were examined from 66 of the 145 persons from whom histories were obtained, and single specimens of vomit from seven. Unfortunately, although specimen jars were handed to many of the employees at the time they were ill, very few specimens were obtained early in the outbreak.

In all cases where specimens were taken the person was either still suffering from diarrhœa, or had suffered within the last day or so. Perhaps the imminence of Christmas detracted from their enthusiasm to provide specimens. Figure 3 shows the results of examination of faecal specimens. The day of illness shown is that on which the first of three specimens was collected from the patient. It happened that the positive specimens were in all cases the first taken from the patients concerned. All samples of vomit were negative.

Of the 66 patients whose stools were examined, 9 showed *Shigella sonnei*—8 factory employees and a child aged 4½ years living nearby. One showed *Shigella flexneri* and one *Salmonella typhi-murium*. The histories of those from whom dysentery bacteria were isolated are summarized as follows :—

Two became ill on the 19th, five on the 20th, two on the 21st and one on the 27th. Of the nine Sonne dysentery cases, four started with vomiting, quickly followed by diarrhoea. Five had diarrhoea only. None passed blood. The diarrhoea was persistent in all cases, as is shown by their having to stay away from work for 3–5 weeks, although they were allowed to return when clinically cured, bacteriological cure not being required. The patient suffering from Flexner dysentery was also away from work for 5 weeks.

The patient from whom *Salmonella typhi-murium* was isolated was a canteen worker who had an attack of diarrhoea lasting a day and a half, beginning on the 22nd. Three daily consecutive specimens, the first on 28th December, were obtained and, as they were negative, she returned to work. Diarrhoea continued on and off, and a stool taken on 14th February, 1951, showed *Salmonella typhi-murium*. Stool specimens from other members of the household were negative.

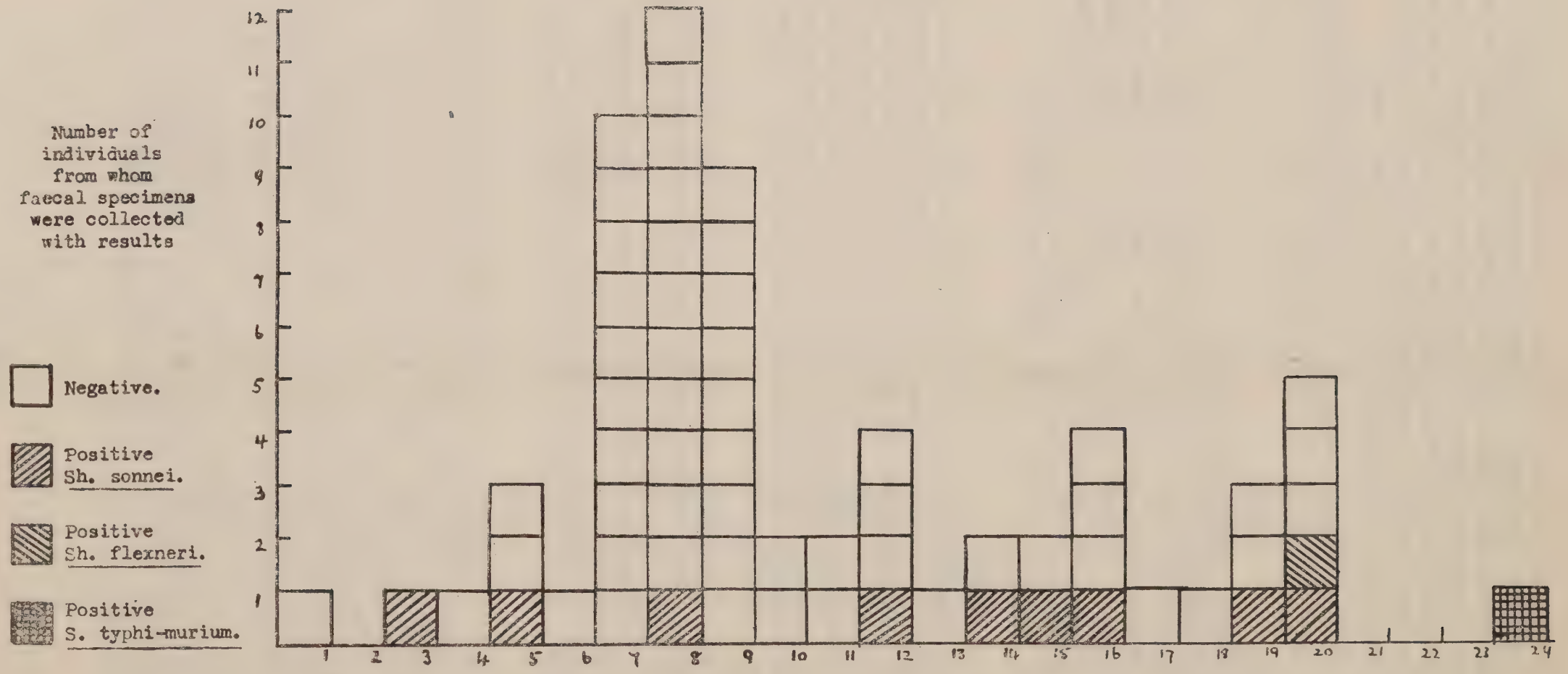


FIG 3. Results of examination of faecal specimens.

The histories of the 145 patients interviewed are summarized in Table 2.

Apart from the positive cases, 121 developed symptoms within two and a half days of the river water being turned on ; these were almost certainly due to infection from the polluted water. Although most patients were ill for only a few days, 41 had diarrhœa for over a week, and 4 of these for over a fortnight. Two patients had diarrhœa lasting over four weeks. Both were admitted to hospital but a specific cause for the diarrhœa could not be found.

Sonne dysentery had been epidemic in Leicester since the beginning of August, 1950, the peak number of 196 notified cases being reached in the week ending 16th December, 1950. During the 4 weeks before the gastro-enteritis started in the works there had only been one person absent from the works as a result of diarrhœa. For 6 weeks before the outbreak there had not been any cases of diarrhœa in the households of the dysentery patients presumably infected by drinking river water.

Sources of pollution of the factory and river water supply

The factory draws its river water from a stream which rises in fields about three miles to the south-west of the works. Just outside the city boundary this stream receives in wet weather the overflow from a small sewer. At the works in addition to the stream there is the River Soar, which also acts as a canal. When in flood, water from the river flows over a weir into the stream, which is 3 ft. below the river. Within the factory itself there is a communication between the river and stream through which a constant trickle of water passes (Fig. 2). The River Soar carries the effluent from several small sewage works situated just outside the City boundary. The effluent from one works goes into a stream which empties into the river opposite the factory. The source of the works' river water is clearly considerably polluted.

Results of bacteriological examinations

Soon after the outbreak of gastro-enteritis was reported a sample of water taken in the canteen on 20th December gave a presumptive coliform count of 180+ per 100 ml. after overnight incubation. On this evidence further inquiries were made and chemical analysis of the factory water and river water performed. The fæcal coli count was later confirmed as 180+ per 100 ml. On direct plating and enrichment through selenite and tetrathionate broth no salmonella or shigella organisms were isolated.

Samples of fæces submitted from those who co-operated were examined for salmonella and shigella bacteria by plating on deoxycholate citrate plates and Wilson and Blair plates direct, and after enrichment through tetrathionate broth. When *Shigella sonnei* was isolated from 9 cases, and *Shigella flexneri* and *Salmonella typhi-murium* from 1 case each, it was decided to make further attempts at isolating *Shigella* from the river water.

Four water samples were taken on the 3rd and 4th January. Three 100 ml. samples were added to equal volumes of double-strength selenite and one to liquid deoxycholate citrate broth. Subcultures were made on to deoxycholate citrate and Wilson and Blair plates after 1, 2 and 3 days' incubation, and representative suspicious colonies picked and identified biochemically and serologically, but no salmonellæ or shigellæ were isolated. Numerous colonies of *Proteus and Ps.pyocyanea* grew on the plates, possibly masking the presence of any pathogens.

Two "Moore" swabs (Moore, 1948, 1950) were left in the river for 24 hours and then transferred to bottles of selenite broth and liquid deoxycholate citrate and subcultures made at 1, 2 and 3 days as previously, but with negative results.

TABLE 2

Summary of Histories of 135 cases of Gastro-enteritis and 10 cases of Dysentery

	Number of Cases	Date in December when first ill									Vomiting Present	Diarrhoea Present	Number Off Work	Number of patients from whom stool specimens taken
		19	20	21	22	23	24	25	26	27				
Cases of Gastro-enteritis occurring among dwellers near factory — proved dysentery cases excluded	27	3	15	7	—	—	—	2	—	—	24	25	4	3
Cases of Gastro-enteritis occurring among factory workers — proved dysentery cases excluded ...	108	21	39	27	9	—	1	2	5	4	73	98	45	53
Total cases of Gastro-enteritis — proved dysentery cases excluded ...	135	24	54	34	9	—	1	4	5	4	97	123	49	56
Sonne Dysentery cases ...	9	1	5	2	—	—	—	—	—	1	4	9	8	9
Flexner Dysentery cases ...	1	1	—	—	—	—	—	—	—	—	1	1	1	1

On two occasions during the next two weeks two Moore swabs were left for 24 hours in a chamber of the factory pumping station where the water was pumped from the river and later filtered and pumped through the factory. The water in these chambers was very turbulent and the swabs may have been washed instead of acting as absorbent filters.

At that time a method was described by Lendon and Mackenzie (1951) for the detection of *Salmonella typhi* in sewage and river water whereby dilution of the enrichment fluid gave better results. As we were not convinced that selenite broth was a satisfactory enrichment fluid for *Shigella sonnei* it was decided to add one swab to peptone water, another to selenite broth and a third, if obtained, to liquid deoxycholate citrate medium. Accordingly on 22nd January two swabs were left for a week in the river in midstream attached to a bridge just above where the water was pumped into the factory. On collection the swabs were placed in jars containing peptone water and selenite broth, and on arrival at the laboratory the fluids were diluted 1/10, 1/100, 1/1,000 in 10 ml. quantities of peptone water and selenite broth respectively. The original fluids and the dilutions were incubated at 37°C. and subcultured at 24 and 72 hours on to deoxycholate citrate and Wilson and Blair plates. From the 1/1,000 peptone water dilution plated after 72 hours on to deoxycholate citrate agar, *Salmonella typhi-murium* and *Shigella sonnei* were isolated. From the 1/1000 selenite broth dilution plated after 72 hours, only *Shigella sonnei* was isolated.

Two weeks later, 5th February, two other swabs were suspended in the same place and *Shigella sonnei* was isolated from the 1/100 peptone water dilution plated after 24 hours. On this occasion no subcultures were made on to Wilson and Blair plates.

We had thus been able to isolate *Shigella sonnei* on two occasions from the river water just above the place where the water was pumped into the factory.

Shortly before our successful isolation of *Shigella* from the river we thought we ought to test the reliability of our methods on material where *Shigella sonnei* was likely to be present, namely the city sewage. Accordingly on 31st January two swabs were left 5 days in one of the main sewers at a point just proximal to the pumping station for the sewage farms. *Shigella sonnei* was isolated from the 1/10 and 1/100 selenite broth dilutions. Later by the same methods *Shigella sonnei* was also isolated at the city sewage farm from the effluent of old filters, and from the outfall entering a stream after this effluent had percolated through the irrigation fields. *Shigella* was not isolated from the effluent or the outfall from the newest and more efficient filters.

In all these laboratory examinations it was apparent that the more non-lactose-fermenting colonies on deoxycholate citrate plates were picked the better was the chance of isolating *Shigella.* For this reason the search for *Salmonella* on Wilson and Blair plates was discontinued so that more colonies could be picked from the deoxycholate plates. Although by our methods we were able to isolate *Shigella,* it was felt that the methods we used were far from ideal. Peso, Leiguarda and Kempny (1949) devised a method of coagulation and precipitation of bacteria from river water samples by the use of aluminium hydroxide, but when they used it in practice on the River Plate numerous salmonellae were isolated but no shigellae.

Discussion on cause of outbreak

It seems probable that most of the cases of gastro-enteritis were due to non-specific causes. Of the 66 patients whose faeces were examined, pathogens were isolated from only 11. Even though many specimens were

not received until a week after the onset of diarrhoea, if all the patients had suffered from a shigella infection one would have expected a higher incidence of positive specimens, as was found in the recent Sonne dysentery outbreak in Leicester. The sudden vomiting was in keeping with a possible toxic or chemical food poisoning. Chemical analysis did not reveal any substance in sufficient concentration to cause poisoning. On the other hand the water was heavily polluted by sewage effluents. There were obviously sufficient bacteria present capable of producing preformed toxins which could have caused the acute symptoms. However, many patients complained of diarrhoea for over a week, and a few for 2 to 4 weeks. It was possible that they had been infected by an organism or organisms which could not be detected by the usual laboratory methods, or which are normally regarded as non-pathogenic. Two patients were admitted to hospital, and in spite of repeated examinations no specific cause could be found. One must assume, therefore, that the gastro-enteritis was probably due to unidentified bacteria or their toxins.

Those who suffered from Sonne dysentery, with the exception of the patient who became ill on the 27th, were almost certainly infected by drinking the contaminated water. In favour of this view are:

1. River water had been distributed throughout the factory drinking water pipes for 52 hours on 18th, 19th and 20th December. This water was known to be contaminated by sewage effluent. *Shigella sonnei* was later isolated from the river water on 22nd January.

2. Careful inquiry among the patients and their families showed that none of them had suffered from gastro-enteritis for 6 weeks before the patients became ill. There was no other obvious source of infection.

3. The illness began explosively 1-2 days after drinking the polluted water.

4. The chances of 8 out of 56 patients carrying *Shigella sonnei,* unless they were suffering from Sonne dysentery, are small. In support of this are the findings of Mackenzie (1951) during the past twelve years when examining Metropolitan Water Board employees whose duties might lead to contamination of the water or who had recently had gastro-enteritis. No pathogenic organisms were found in the stools of 450 healthy employees who had given suspicious agglutination reactions with typhoid or para-typhoid bacilli. Of 1,150 employees who had suffered from gastro-enteritis, salmonellae were isolated from 10 and *Shigella sonnei* from 9.

Against the view that the dysentery cases were infected by river water is the fact that there had been a considerable number of Sonne dysentery cases in Leicester immediately preceding the outbreak, and it may be argued that stool specimens of 56 persons selected at random would have produced similar results.

We consider that the weight of evidence is in favour of the eight patients having been infected by drinking the contaminated river water.

Another Outbreak of Gastro-enteritis

Two weeks after the outbreak we were informed of an outbreak of gastro-enteritis in a works just across the canal from the factory where the water-borne gastro-enteritis had occurred. The water supplies came from entirely different mains. On arrival at the works to investigate the cause of the out-break, we were shown a sample of turbid water said to have been drawn from a tap in the canteen. Inquiry showed that there was a cross-connection in the works, closed by a valve, between the town mains and the river supply.

Unfortunately, to spoil the story, further inquiry indicated that the outbreak was probably due to contaminated gravy. The turbid water had been taken from a tap which had not been in use for a very long time, the turbidity being due to excess iron. The results of the chemical and bacteriological analysis were otherwise within normal limits. Water samples from other parts of the works were also normal. The valve in the connecting pipe was firmly closed and leakage was impossible. However the City Water Engineer asked for the cross-connection to be removed.

Previous Outbreaks of Water-borne Dysentery

Outbreaks of water-borne Sonne dysentery are rare in this country. The only one which we have been able to trace where the evidence was conclusive was that reported by Green and Macleod (1943). Four hundred persons out of a population of 10,000 in a Somerset town were infected by drinking contaminated, inadequately chlorinated water derived from deep wells. *Shigella sonnei* was isolated from the stools of the infected persons and also from the water itself.

Another outbreak which was considered to be spread by water was reported by Nisbet (1938) in Kilmarnock. On reviewing the evidence in support of water-borne infection, we do not consider it conclusive. The outbreak was not explosive, the 162 persons affected becoming ill between 14th August, 1937, and 1st June, 1938. At that time it was concluded that the water contained *Shigella sonnei* because of a positive bacteriophage test. This test is not now regarded as specific, the reaction being given by *Bact. coli* and other non-pathogenic bacteria commonly found in water.

Several water-borne outbreaks have occurred in the United States of America. Kinnaman and Beelman (1944) reported an outbreak of 3,000 cases of dysentery in Kansas State, after admission of sewage from pits under water closets to water mains through hydrant valves. The predominant organism was *Shigella flexneri.* Eliassen and Cummings (1948) give an account of water-borne outbreaks of illness between 1938 and 1945. Of the 327 outbreaks resulting in 111,320 cases, 35 outbreaks and 8,622 cases were due to dysentery. Cross-connections with a polluted water supply accounted for 3 outbreaks of dysentery—3,000 cases in all.

Details of a dysentery outbreak in Germany caused by polluted well water are given by Müller (1947). The wall of the well, which was 4 metres deep, was formed of pebbles. Latrines were situated 6 metres away and percolation from the latrines into the well could occur. Organisms of the dysentery group, Type L (*Shigella flexneri* Type 6), were isolated from both the infected persons and the well water. Sorvina (1946) reported, in a town in the Ukraine, an epidemic which he considered to have been caused by sewage contamination of a piped water supply, but *Shigella sonnei* was not demonstrated in the water.

It is interesting also to mention the Chicago epidemic of amoebic dysentery of 1933 (Report 1933) where the two major points of pollution which resulted in water-borne infection in two hotels were (*a*) two cross-connections between an overhead sewer and a water pipe, and (*b*) an old rotting wooden plug in an overhead sewer which permitted leakage into drinking water tanks below.

The Legal Position

Of the two cross-connections noted in this paper one was of many years' standing, the other recent.

Since water-borne outbreaks may occur resulting from cross-connections between a pipe containing purified water and a pipe containing untreated water, we think it will be useful to state the legal position.

Cross-connections were first prohibited by the Waterworks Clauses Act, 1863. This Act was incorporated in the Leicester Waterworks Act of 1866, which is still operative in the City. The Waterworks Clauses Act, 1863, is repealed by the Water Act, 1945, S.62. The Model Byelaws published with the Water Act, 1945, prohibit cross-connections. Local authorities supplying water have either adopted similar Byelaws or have local Acts, as at Leicester.

It is evident, therefore, that cross-connections are prohibited.

Summary

1. An account is given of an outbreak of water-borne gastro-enteritis and Sonne dysentery occurring among employees of a factory.

2. Altogether 160 employees and 28 persons living in neighbouring houses were affected. There were 9 cases of infection by *Shigella sonnei,* 1 by *Salmonella typhi-murium,* and 1 by *Shigella flexneri.*

3. The cause of the outbreak was the admission of crude river water to the town mains through a cross-connection in the factory between two pipes, one carrying river water at 240 lb. pressure, the other carrying town water at 25-30 lb. pressure.

4. *Shigella sonnei* was later isolated from the river water.

Our thanks are due to Dr. E. K. Macdonald, Medical Officer of Health, Leicester, for help and advice throughout the inquiry and for criticism of this report; to Mr. F. C. Bullock for the reports on the chemical analysis of water samples and for advice and criticism; to Mr. T. S. Griffin, Water Engineer and Manager of the City of Leicester Water Department, to Mr. P. D. Murphy, his deputy, and Mr. T. G. Morris, Mr. W. R. Keily, and Mr. K. Rodwell of that Department, for help during the inquiry and for the preparation of the plan of the factory; to the sanitary inspectors of the Leicester Health Department, especially Mr. A. Welton and Mr. J. Hawkesley, for their help; to the laboratory technicians of the Public Health Laboratory Service, Leicester, for the numerous examinations they undertook; and to Miss J. Carter-Brown and Mr. M. A. Yeomans of the Leicester Health Department who prepared Figures 2 and 3.

References

Eliassen, R. and Cummings, R. H. (1948) *J. Amer. Wat. Wks. Ass.*, **40**, 509.

Glover, J. A. (1947) *This Bulletin*, **6**, 46; (1949) *Ibid.*, **8**, 138.

Green, C. A. and Macleod, M.C. (1943) *Brit. med. J.*, ii, 259.

Hobbs, B. C. and Allison, V. D. (1942) *This Bulletin*, **1**, 1.

Kinnaman, C. H. and Beelman, F. C. (1944) *Amer. J. publ. Hlth.*, **44**, 948.

Lendon, N. C. and Mackenzie, R. D. (1951) *This Bulletin*, **10**, 23.

Mackenzie, E. F. W. (1951) *Publ. Hlth.*, **64**, 47.

Moore, B. (1948) *This Bulletin*, **7**, 241; (1950) *Ibid.*, **9**, 72.

Müller, G. (1947) *Zbl. Bakt.*, **152**, 133.

Nisbet, B. R. (1938) *Med. Offr.*, **59**, 87.

Peso, O. A., Leiguarda, R. H. and Kempny, J. C. (1949) *Rev. Obr. san. Nación.*, **13**, 101.

Report (1933) *Amer. Nat. Inst. Hlth., Wash.*, Bull. No. 166.

Sorvina, L. E. (1946) *Vrach. delo.*, **26**, 743.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health, (Room 207),
Savile Row,
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The January Bulletin was issued on 25th January.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(14455) Wt. 8055—6219 3300 2/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

MARCH **1952** **Vol. 11**

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the
Ministry of Health, Savile Row, W.1.

RECENT TRENDS OF DIPHTHERIA

W. P. D. Logan, M.D., Ph.D., D.P.H., Chief Medical Statistician,
General Register Office, W.C.2.

Almost 10,000 persons died from diphtheria in England and Wales in 1901 ; 32 in 1951 (provisional figure). From being one of the most serious causes of death of children in this country, diphtheria has now fallen to a position of numerical insignificance.

It is to be noted that of the total reduction in the mortality from diphtheria during the past half century by far the greatest part has taken place in the last 10 years, the period of large scale immunisation ; and that during these 10 years there has been a sudden and enormous reduction in the incidence of notified cases as well as of deaths.

The situation is now being reached—a situation scarcely dreamed of in 1940 when the immunisation campaign started—where the eradication of diphtheria as an indigenous disease in this country can be foreseen as a very real possibility within the next few years, providing there is no slackening in the immunisation efforts that have been so dramatically successful in the past 10 years. Complacency resulting from what has already been achieved, or loss of interest or of confidence in immunisation, may mean that diphtheria will go on occurring endemically and epidemically in this country indefinitely, with the ever-present risk of a return to high mortality ; but a vigorously continued immunisation programme, combined with existing methods of epidemic control, may free us entirely from the disease except for the occasionally imported case.

Already diphtheria is becoming an exceptional occurrence even in types of locality where formerly its incidence and mortality were particularly high. There are 83 county boroughs in England and Wales. Table I shows the rapidly increasing number of these county boroughs that had no deaths from diphtheria and no final (i.e. confirmed) notifications* of diphtheria during successive years 1946 to 1950.

TABLE 1

Diphtheria: Numbers of County Boroughs with no deaths and no final notifications*, 1946 *to* 1950

	Numbers of County Boroughs	
	With no deaths	With no final notifications
1946	28	—
1947	36	3
1948	56	7
1949	63	12
1950	70	33

70 county boroughs had no deaths from diphtheria in 1950, and there were 14 county boroughs, with an aggregated population approaching one and a quarter million persons, that did not have a single death from diphtheria during the whole five-year period, viz. Blackburn, Blackpool, Canterbury,

* Final notifications are notifications corrected for subsequent amendments of diagnosis by the notifying practitioner or by the medical superintendent of the infectious diseases hospital.

Chester, Dewsbury, Eastbourne, Hastings, Oxford, Preston, Southend, Southport Warrington, Worcester, York.

No county boroughs succeeded in being entirely free of confirmed cases throughout the whole five years, but Canterbury had no cases during the four years 1947 to 1950, and Derby and Southend had none during the three years 1948 to 1950.

TABLE 2

Diphtheria: Notifications (Original and Final), Deaths and Fatality Rates. England and Wales, 1944 to 1951

	Notifications				Deaths		Fatality (Deaths per cent. of Notifications)	
	Original		Final					
	No.	Rate per million	No.	Rate per million	No.	Rate per million	Original	Final
1944	28,674	759	22,085	584	908	24·0	3·2	4·1
1945	24,077	631	17,580	461	694	18·2	2·9	3·9
1946	17,823	439	11,601	286	455	11·2	2·6	3·9
1947	10,315	247	5,492	131	242	5·8	2·3	4·4
1948	7,973	187	3,531	83	155	3·6	1·9	4·4
1949	4,960	115	1,874	43	84	1·9	1·7	4·5
1950	2,829	65	959	22	49	1·1	1·7	5·1
1951	1,983	45	699	16	32	0·73	1·6	4·6

Recent trends of rates

Table 2 gives notification, death, and fatality rates from 1944 and indicates little slackening as yet in the rapid downward trend of cases and of deaths.

TABLE 3

Diphtheria: Notification (Final), Death, and Fatality Rates among Immunised and Non-immunised Children aged under 15. England and Wales, 1945–1947

	Immunised			Non-Immunised		
	Final Notification Rate per 100,000	Death Rate per million	Fatality Rate (deaths per 100 notifications)	Final Notification Rate per 100,000	Death Rate per million	Fatality Rate (deaths per 100 notifications)
1945...	82	7	0·9	266	172	6·5
1946...	33	3	0·8	87	45	5·2
1947...	18	2	1·1	52	44	8·5

Table 3 compares notification, death and fatality rates of children under 15 according to whether or not the children had been immunised. Over the three years covered in the table the notification rate was 3 times higher, the death rate 22 times higher, and the fatality rate 7 times higher among non-immunised than among immunised children.

Although the value of immunisation is beyond serious dispute some of the credit for the recent trends should perhaps be accorded to *Corynebacterium diphtheriae* itself. For instance, table 4, based on figures previously published in reports from the Public Health Laboratory Service (this Bulletin 1945, Vol. IV, page 152 ; and 1949, Vol. VIII, page 116), shows that the proportion

of *mitis* strains recovered between 1945 and 1948 almost doubled, whereas the proportions of *intermedius* and *gravis* substantially declined. Table 2 has shown, however, that there has been no great reduction in fatality—in fact the ratio of deaths to *final* notifications has tended slightly to increase.

TABLE 4

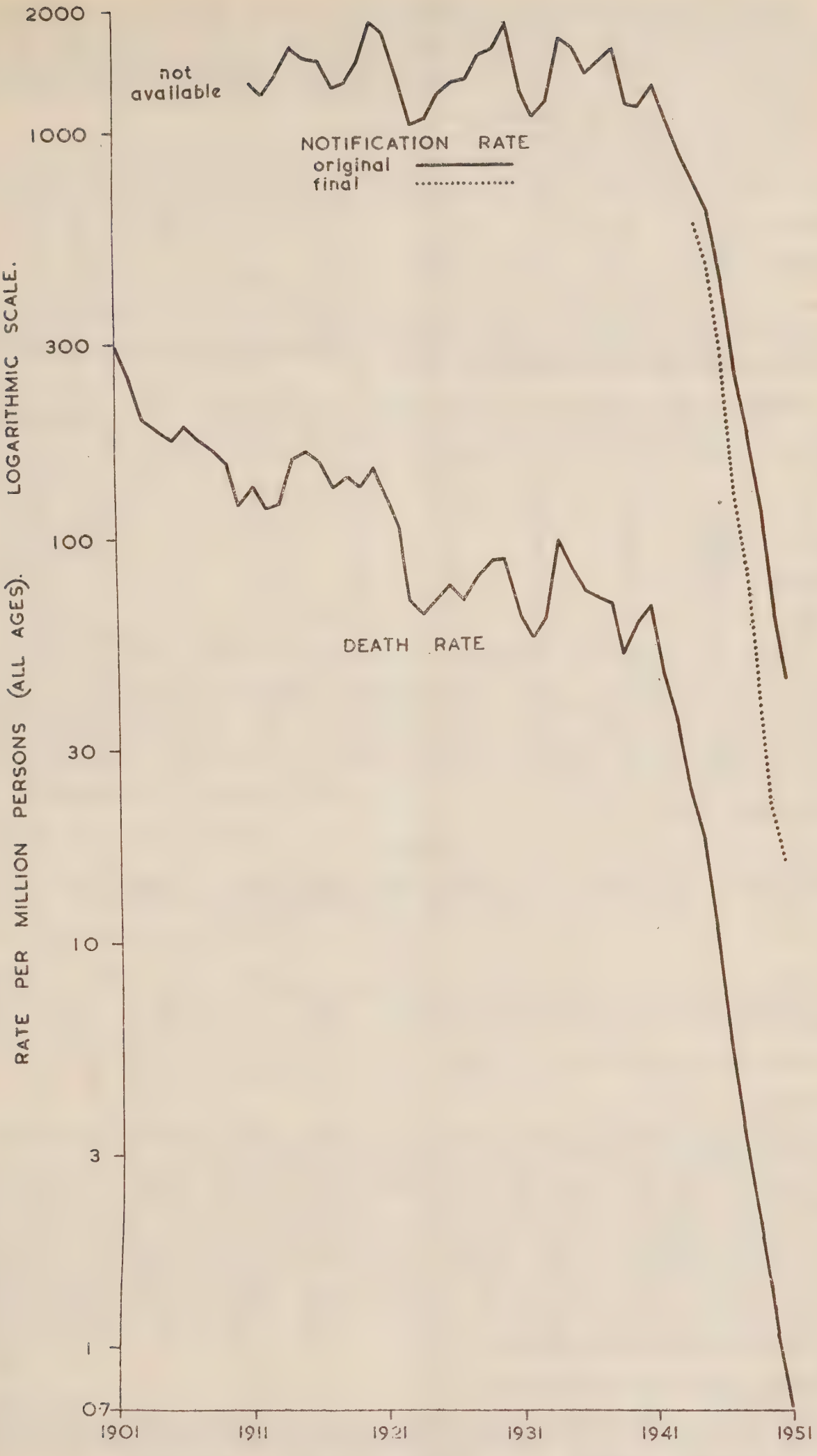
Percentage Distribution of Diphtheria Types

	MITIS	INTERMEDIUS	GRAVIS	INDETERMINATE	TOTAL	NO.
1942 ...	25	29	45	1	100	9892
1943 ...	25	30	44	1	100	6981
1944 ...	21	29	48	2	100	5978
1945 ...	28	28	39	5	100	5051
1946 ...	31	20	35	14	100	4048
1947 ...	40	14	31	15	100	1915
1948 ...	52	10	24	15	100	1195

TABLE 5

Diphtheria: Notification (Final) and Death Rates by Sex and Age: England and Wales, 1944 *to* 1950

	Age							
	0—	1—	3—	5—	10—	15—	25 and over	All Ages
Notification Rates per million:								
Male								
1944	476	1,219	2,341	2,480	1,429	632	52	583
1945	301	912	1,878	2,004	1,128	526	43	462
1946	281	717	1,176	1,227	679	281	44	274
1947	123	422	644	577	262	122	20·5	128
1948	70	211	396	341	187	70	12·4	77
1949	53	117	208	180	107	28	7·8	42
1950	34	92	104	88	43	17·3	4·2	22
Female								
1944	333	1,013	2,283	2,655	1,726	893	133	586
1945	248	745	1,716	2,214	1,382	673	105	460
1946	179	543	1,083	1,239	902	459	77	296
1947	82	280	528	563	376	201	37	135
1948	50	170	335	366	247	132	23·7	88
1949	31	102	186	171	128	59	13·6	45
1950	20	50	73	73	61	33	6·8	22
Death Rates per million								
Male								
1944	53·8	97·4	158·1	119·6	40·6	12·0	1·4	26·7
1945	33·8	92·1	113·6	86·6	28·3	10·5	2·1	20·9
1946	36·1	66·5	61·4	39·7	14·6	4·6	1·8	11·0
1947	20·1	30·5	37·2	29·1	9·3	2·3	0·7	6·3
1948	10·0	25·4	23·4	15·6	6·4	0·4	0·4	3·8
1949	2·7	7·1	23·5	7·2	0·7	0·8	0·6	2·2
1950	—	6·5	7·7	4·5	2·8	1·4	0·4	1·5
Female								
1944	20·8	110·1	203·3	122·3	32·9	8·9	3·4	22·0
1945	17·9	80·7	102·0	94·6	24·7	6·8	3·5	16·1
1946	29·3	59·3	77·6	49·7	9·7	8·3	3·3	11·4
1947	16·5	30·7	34·4	28·0	6·6	2·4	0·9	5·3
1948	5·2	12·0	21·5	22·6	3·7	1·4	0·7	3·4
1949	2·8	12·5	13·7	4·8	3·6	—	0·5	1·7
1950	—	4·1	6·7	2·7	0·7	0·3	0·3	0·8



Diphtheria Notification and Death Rates.
England and Wales 1901–1951.

14860

Table 5 gives the trends of notification and death rates by *sex* and *age* from 1944 to 1950. The notification rates for males and females at "all ages" in 1950 were 4 per cent. of the rates in 1944. At separate ages the notification rates in 1949 and 1950 expressed per cent. of corresponding rates in 1944 were as follows:—

		0—	1—	3—	5—	10—	15—	25+	All Ages
Males	1949	11	10	9	7	7	4	15	7
	1950	7	8	4	4	3	3	8	4
Females	1949	9	10	8	6	7	7	10	8
	1950	6	5	3	3	4	4	5	4

Improvement tended to be very slightly less among the youngest children and among adults than at intermediate ages.

Similarly the death rates at separate ages in 1949 and 1950, expressed per cent. of corresponding rates in 1944, were as follows:—

		0—	1—	3—	5—	10—	15—	25+	All Ages
Males	1949	5	7	15	6	2	7	43	8
	1950	0	7	5	4	7	12	29	6
Females	1949	13	11	7	4	11	0	15	8
	1950	0	4	3	2	2	3	9	4

There were no deaths under one year of age in 1950. Owing to the small number of deaths now occurring at separate ages the proportionate changes tend to be somewhat erratic. It is evident, however, that the smallest relative improvement has taken place among the adult males, and as a result the death rate of males age 25 and over which in 1944 was 5 per cent. of the male rate at "all ages," had increased relatively by 1950 and was 27 per cent. of the "all ages" rate.

TABLE 6

Diphtheria: Sex Ratio. Notification and Death Rates of Males per cent. of Females. Average for 1944–1950

	0—	1—	3—	5—	10—	15—	25+	All Ages
Notification rates ...	142	127	109	95	80	68	46	97
Death rates	169	105	93	93	125	114	59	119

Sex ratio

Table 6 compares the notification and death rates of males with those of females during the combined period 1944-1950, the male rates at separate ages being expressed per cent. of the corresponding female rates. Notifications were more frequent among boys than among girls under five, but at adult ages the female rate was double that of males. The death rate of male infants was much higher than that of female infants, and there was also a male excess at ages from 10 to 24. At "all ages" there was a slight excess (3 per cent.) of notifications of females, but a much larger excess (19 per cent.) of deaths among males.

Original and Final Notifications

Since 1944 arrangements have been in force for the reporting of amendments of diagnosis made either by the notifying practitioner or by the infectious diseases hospital.

In 1944 the proportion of final notifications of diphtheria, i.e. after revision of diagnosis, was 77 per cent. of original notifications. This proportion has declined each year since, and by 1950 final notifications were only 34 per cent. of the number originally notified.

TABLE 7

Diphtheria: Original and Final Notifications, England and Wales; Final Notifications per cent. of Original, England and Wales, Standard Regions and Density Aggregates, 1944 *to* 1950

	1944	1945	1946	1947	1948	1949	1950
England and Wales:							
Original No. ...	29,949	25,246	18,283	10,465	8,035	4,982	2,832
Final No.	23,152	18,571	11,967	5,592	3,560	1,881	959
Final per cent. of Original	77	74	65	53	44	38	34
Standard Regions: Final per cent. of Original							
Northern	89	87	80	61	56	48	43
E. & W. Ridings ...	82	76	69	59	39	37	33
North Western ...	68	64	53	44	40	31	26
North Midland ...	80	78	76	64	59	37	23
Midland	74	68	60	54	43	42	42
Eastern	83	75	76	75	74	77	68
London & South E.	68	69	62	49	41	36	27
Southern	85	85	86	81	76	67	58
South Western ...	78	75	64	53	50	31	41
Wales	76	75	73	59	36	37	43
Greater London ...	65	64	58	46	38	33	25
County Boroughs ...	72	66	57	45	37	32	31
Other Urban Districts	84	81	77	67	57	49	42
Rural Districts ...	87	87	78	68	58	50	46

Table 7, which expresses final notifications per cent. of original from 1944 to 1950 in the country as a whole and in the various geographical regions and density aggregates, indicates that the proportions of final to original notifications have declined in all areas. Corrections of diagnosis

TABLE 8

Diphtheria: Original and Final Notification: Final per cent. of Original. Regions by Classes of Area. England and Wales, 1949

	County Boroughs*			Other Urban Districts*			Rural Districts		
	Original	Final	Final per cent. of Original	Original	Final	Final per cent. of Original	Original	Final	Final per cent. of Original
South East*	75	34	**45**	872	325	**37**	59	42	**71**
North ...	1,652	523	**32**	597	271	**45**	223	88	**39**
Midland ...	688	211	**31**	254	148	**58**	66	30	**45**
East ...	37	26	**70**	29	8	**28**	18	14	**78**
South West	70	30	**43**	26	11	**42**	21	11	**52**
Wales ...	77	18	**23**	138	46	**33**	58	38	**66**
England and Wales* ...	2,599	842	32	1,916	809	**42**	445	223	**50**

* Including Greater London.

14860 A 3

have been reported most frequently in London and the large towns, less in the smaller towns, and least in the rural districts. It should be mentioned that some of the other infectious diseases tend to show a similar type of urban-rural differentiation, but not to the same extent as diphtheria.

The proportions of original and final notifications in 1949 are analysed further in Table 8 by urban and rural areas within the main regions; and Table 9 gives similar details for the 25 largest towns in England and Wales, arranged in alphabetical order.

TABLE 9

Diphtheria: Original and Final Notifications; and Final Notifications per cent. of Original: 25 large towns in England and Wales, 1949.

	Original	Final	Final per cent. of Original
Birkenhead	182	80	**44**
Birmingham	455	138	**30**
Bolton	32	8	**25**
Bradford	20	20	**100**
Bristol	71	4	**6**
Cardiff	38	1	**3**
Coventry	17	12	**71**
Croydon	3	2	**67**
Kingston-upon-Hull	38	2	**5**
Leeds	44	5	**11**
Leicester	16	6	**38**
Liverpool	568	129	**23**
London A.C.	592	219	**37**
Manchester	135	22	**16**
Newcastle-on-Tyne	52	4	**8**
Nottingham	21	1	**5**
Plymouth	66	30	**45**
Portsmouth	8	7	**88**
Salford	41	2	**5**
Sheffield	54	3	**6**
Southampton	2	1	**50**
Stoke-on-Trent	13	3	**23**
Sunderland	18	16	**89**
Swansea	3	—	**0**
West Ham	21	4	**19**

In several towns the proportion of final notifications was remarkably low, for instance, 1 out of 38 in Cardiff, 2 out of 38 in Kingston-upon-Hull, 1 out of 21 in Nottingham, 2 out of 41 in Salford, and 3 out of 54 in Sheffield. In sharp contrast the proportions were 20 out of 20 in Bradford, 16 out of 18 in Sunderland and 7 out of 8 in Portsmouth. Aggregating the 25 towns there were 719 final notifications, or 29 per cent. of the 2,510 cases originally notified.

It is evident that in many areas, chiefly the large towns, the number of cases originally notified as diphtheria now bears little relationship to the number of cases in which the diagnosis can be afterwards confirmed. It may be that practitioners are tending more and more to notify on suspicion, and with a formidable disease like diphtheria this is a reasonable course to take. On the other hand hospitals may be becoming increasingly reluctant to confirm a clinical diagnosis of diphtheria unless *C. diphtheriae* can be definitely recovered.

DEATHS FROM RHEUMATOID ARTHRITIS 1940-1949

Eileen M. Brooke, M.Sc., Statistician, General Register Office.

From 1939 onwards the Registrar General has distinguished deaths from rheumatoid arthritis and its synonyms from those due to other forms of arthritis. Some statistics of deaths from rheumatoid arthritis for the decade 1940-1949, classified according to the Fifth Revision of the International List, are presented below.

TABLE 1

Numbers of Deaths attributed to Rheumatoid Arthritis 1940-1949. (*Excluding non-civilian males, and non-civilian females on and after 1st June,* 1941.)

Sex	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Males	331	365	376	321	320	321	288	287	247	286
Females	1,122	1,002	916	953	895	945	856	834	854	910

In all 3,142 males and 9,287 females were stated to have died from rheumatoid arthritis in the ten years; the annual numbers of deaths are shown in Table 1.

Table 2 shows the crude death rates at all ages. Rates of men rose from 18 per million living in 1940 to 22 in 1942, and after remaining at 20 for three years, showed a downward trend to 12 per million in 1948, followed by an increase in 1949. Rates for women fell from 52 per million in 1940 to 43 in 1942, oscillated during 1943-45, and after falling to 38 per million in 1948 increased to 40 in 1949. (Fig. 1.)

TABLE 2

Rheumatoid Arthritis. Crude Death Rates at all Ages, per Million living, 1940-49. (*Excluding non-civilian males, and non-civilian females on and after 1st June,* 1941.)

Sex	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Males	18	21	22	20	20	20	15	15	12	14
Females	52	47	43	44	41	43	39	38	38	40

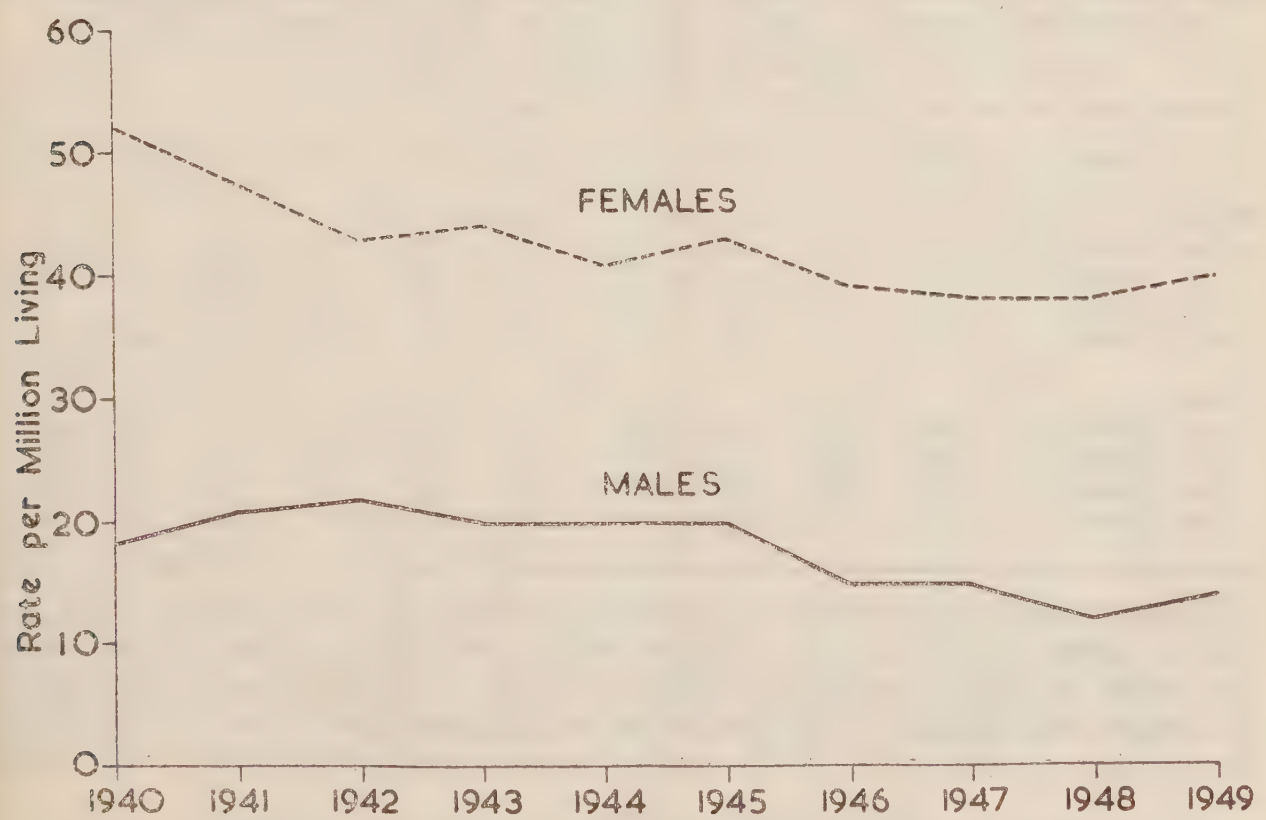


Fig. 1. Rheumatoid Arthritis. Crude Death Rates at all ages per Million living.

TABLE 3

Rheumatoid Arthritis. Death-rates by Sex and Age. 1940-1949.

Year	Death Rates per Million living*											
	MALES						FEMALES					
	0—	25—	45—	55—	65—	75 and over	0—	25—	45—	55—	65—	75 and over
1940	0·6	2·2	12·0	43	95	200	0·5	3·4	27·0	103	255	590
1941	0·5	1·9	10·8	40	105	272	0·3	5·6	25·1	75	242	504
1942	0·3	3·2	10·7	47	102	239	0·9	3·3	23·9	75	201	455
1943	0·7	1·7	8·2	35	88	214	0·5	4·6	18·8	78	217	429
1944	0·7	1·7	12·1	35	82	194	0·7	3·9	22·7	68	193	397
1945	0·7	3·2	10·7	39	77	165	0·7	5·1	21·5	66	192	431
1946	0·5	2·1	7·9	29	81	153	0·3	4·0	17·0	68	176	369
1947	0·9	0·8	6·5	34	65	185	0·3	3·0	20·5	52	173	371
1948	0·8	1·7	9·7	22	60	136	—	2·5	13·6	58	177	370
1949	0·4	2·9	6·5	28	76	142	0·3	2·5	17·4	62	171	400

* Including deaths of non-civilians in England and Wales, and based on total population inclusive of armed forces at home and abroad.

It will be seen from Table 3 that there were considerable variations in the death rates for both sexes within each of the six age-groups shown. In each year the rates increased with age, those for women at ages 45 and over increasing a little more sharply than those for men. There was a general downward trend over the ten years in the rates for both sexes at ages 45 and over. The average rates for the years 1947-49 expressed as percentages of those for 1940-42 in the four age groups from 45 upwards were, men 68, 65, 67, 65, women 68, 68, 75 and 74.

TABLE 4

Rheumatoid Arthritis. Deaths by Quarter of Occurrence. 1940-1949.
(*Excluding non-civilian deaths.*)

Quarter	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Males										
1st Quarter ...	109	115	129	79	83	99	90	113	61	97
2nd Quarter ...	74	94	85	93	92	79	65	63	58	70
3rd Quarter ...	52	78	75	64	67	59	60	54	61	57
4th Quarter ...	96	78	88	83	84	79	73	57	67	60
Females										
1st Quarter ...	363	321	289	250	290	311	291	258	209	287
2nd Quarter ...	243	264	214	258	192	236	211	215	204	215
3rd Quarter ...	237	205	200	188	193	187	170	172	208	186
4th Quarter ...	270	213	214	257	221	210	186	192	237	223

Table 4 shows the number of deaths occurring in each quarter of the ten years; these figures do not compare with those in the other tables which are based on deaths registered during the year. For both sexes the number of deaths was highest in the first or second quarters and lowest in the third. In the first quarter of 1948 the deaths were considerably fewer than might

have been expected (see Fig. 2); this was however the mildest first quarter of the decade.

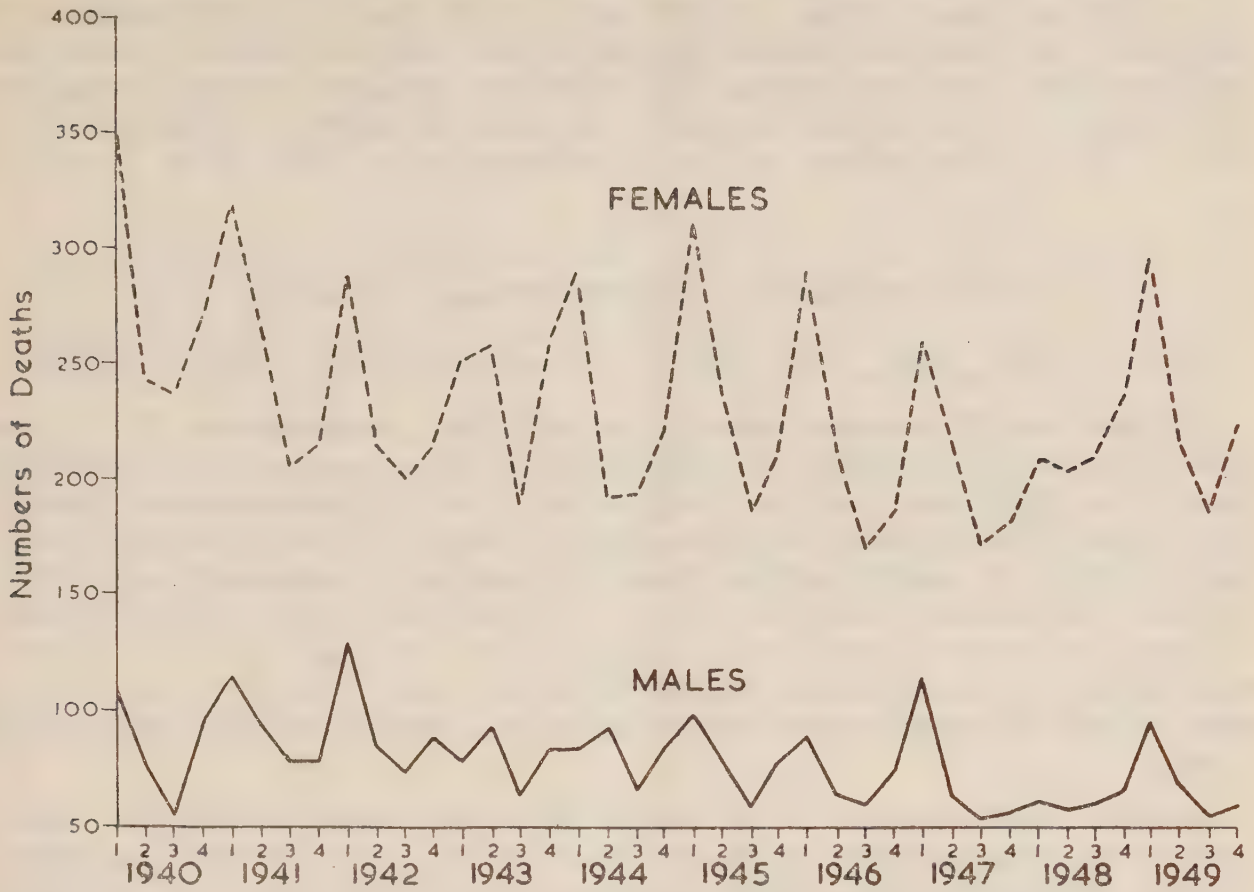


Fig. 2. Rheumatoid Arthritis. Deaths occurring in Quarters, 1940–49.

As rheumatoid arthritis is a cause of death showing a well-marked sex differential, the death rates for males and females at all ages were calculated for 1947-49 by geographic regions. These rates expressed as percentages of the England and Wales rates are shown in Table 5, together with the ratios of the rates for females to those of males.

TABLE 5

Rheumatoid Arthritis. Regional death rates compared with those for England and Wales and ratio of female to male rates in each area. 1947-1949. (*Excluding non-civilian deaths.*)

Area	1947			1948			1949		
	Male	Female	F/M	Male	Female	F/M	Male	Female	F/M
England and Wales ...	100	100	2·6	100	100	3·1	100	100	2·9
Regional Summary									
Greater London ...	45	71	4·0	68	62	3·1	89	71	2·3
Remainder of South East	109	91	2·2	130	101	2·5	94	101	3·2
North I	123	111	2·3	135	92	2·1	74	82	3·3
North II	88	109	3·2	64	133	6·5	104	110	3·1
North III	100	116	3·0	126	97	2·4	78	105	3·9
North IV	94	126	3·5	111	116	3·3	128	126	2·9
Midland I	103	82	2·0	52	86	5·2	93	91	2·9
Midland II	137	78	1·5	64	104	5·1	160	113	2·1
East	127	143	2·9	173	123	2·2	31	98	9·3
South West	121	116	2·5	101	94	2·9	127	110	2·5
Wales I	198	134	1·8	128	164	4·0	90	112	3·6
Wales II	82	136	4·3	143	215	4·7	172	175	3·0

During 1947-49, the death rates in Greater London were less than those for the whole country for both males and females. Rates for males were above the national average in each year in the South Western region, as were those for females in North II and IV, and both the Welsh regions. In Midland I the rates for females were below the national average during the three years. The lowest sex-ratio of rates occurred in Midland II in 1948 (1·5) and the highest in the Eastern region in 1949 (9·3).

THE PRESENT NUTRITIONAL STATE, I

W. T. C. Berry, M.A., M.D., and P. J. Cowin, M.R.C.S., L.R.C.P., D.P.H.

Previous reports on this subject deal with the examinations of a representative sample of children of both sexes in order to assess the nutritional state. Our experience, both in England and in Occupied Germany (Berry, Cowin and Magee, 1951) indicates that the male is a more sensitive indicator of mal- or under-nutrition than the female. Accordingly since 1950 we have confined our attention to boys. Of 1,477 boys aged 6-14 examined, 8·4 per cent. were graded "fair" and 0·5 per cent. as "poor". In previous years the corresponding figures for boys were: 1948, 7·5 and 1·1 per cent.; 1949, 7·7 and 1·4 per cent. There has, therefore, been little or no change.

An issue of importance in the use of the rapid method of clinical assessment is the comparability of results from year to year. On this and other aspects of the procedure a test was arranged for us by Dr. E. R. Bransby, two trials being made at an interval of more than a year. Of the four clinicians who were present at both trials, Bransby and Hammond (1951) report "their standards in relation to each other are the same in both trials".

Even though it appears from this that the comparisons made in these reports from year to year are valid, still we feel that more objective measures are needed in order to detect slight changes occurring gradually over long periods (and this is what is likely to be required in future years). We are therefore collecting base line data on certain readily accessible communities, with which the findings of future years may be compared.

In deciding what tests to use, we have been guided by the experience of workers (including ourselves) in the occupied countries of Europe during and after the war. The common findings were of a general nature; wasting, loss of weight, pallor, slight anaemia, some loss of strength, retardation of growth and development, marked reduction in activity and endurance, and, occasionally, oedema. We have taken the view that nutrition surveys should be designed to detect the earliest signs of these. Specific deficiencies were rare on the continent, and we feel that in England they should be sought only in groups where, for any reason, they might be expected to occur and using methods specifically designed for their detection.

For the first objective, i.e., the collection of baseline data of a general nature, we employ the following tests. (1) A number of anthropometric measurements, (2) haemoglobin level of blood, (3) lumbar pull, (4) measurements of skin-fold thickness, (5) (on a tentative and experimental scale), the level of pseudocholinesterase in the plasma. We have rejected the use of serum protein determinations because our own experience in Berlin and that of many other workers in post-war Europe shows that it is unlikely to be of value at the levels of nutrition which may be expected to prevail in England.

To date we have examined about 1,500 children in this way. The haemoglobin levels of these and of a number of adults, have been reported elsewhere (Berry, Cowin, and Magee, 1951); they show either no change or a slight rise compared with values found by the M.R.C. in 1943. We have arranged in 1952 to continue with the examination of adults. With regard to the children, a full report is in process of compilation. Below is a summary of the main findings:—

(1) There are differences in the physique of different social and geographical groups which seem to be of much the same order as they were before the war (Friend and Bransby, 1947), despite the increase in availability of protective foods to children of lower income groups as a result of school milk or meals, food subsidies, and equality of rations.

(2) In two localities, one in the industrial North-West, the other in a prosperous dormitory town in the South, the average birth weights were found to be the same; yet by the age of 6 a weight difference of about seven per cent. had occurred between the two groups. Thereafter, this difference was maintained between the two up to the age of 17½ years.

TABLE I

Weights and Heights of one locality expressed as a percentage of the other

Age	*Weight*	*Height*
At birth	100	—
6 years	92·4	96·9
10 „	88·0	95·8
14 „	91·3	96·0
17 „	93·2	95·7

Such differences are well known to exist in different parts of Britain and are often considered to be racial in character; yet genetic factors would presumably operate during intrauterine as well as extrauterine life, so that differences would already be apparent at the time of birth, were this the sole or even the main cause. We hope some time to study more fully the question of when differences begin to appear, and what the cause may be.

(3) No constant difference was found in the physique of boys who took school milk, compared with those who did not, or of those who took school meals compared with those who did not. Nor was there any difference between boys, who through faddiness, eschewed meat, fish, cheese, vegetables, milk, or eggs, or any combination of these. This seems to indicate that the home diets of these children were adequate, yet it does not follow that the diets of the other children would have been adequate without school meals and milk. But if the general level of diet in children was unsatisfactory or even border-line in any respect, the omission of these foodstuffs would undoubtedly have been reflected in changes in physique. The findings of Beltram and Bransby (1950) are of interest in this respect. They surveyed the diets of children from the lowest-paid income group (£5 10s. 0d. per week or less) and found their nutrient content adequate on the average as compared with the recommendations of the B.M.A. Committee on Nutrition (1950).

(4) In a 14-year-old group (in collaboration with Miss C. M. Wood, senior dietitian of the Bristol Public Health Service), the association between physique and diet was studied, the latter part of the study being limited to questioning upon the frequency with which certain foodstuffs appeared in the dietary. The findings showed increases in weights and

other physical measurements with increasing frequency in consumption of such foods as meat and fish, or milk, or fruit, but the relationship was not found to be statistically significant. There was also a suggestion that some non-dietetic factor might be partly, if not wholly, responsible; for when children on the same types of diet but of different social class were compared, it was found that those in the higher social classes (using the Registrar-General's system of classification) were heavier than the others. Similarly grammar school boys were heavier than modern school boys even when there was apparently no difference in the diet.

(5) At least one non-dietetic factor was associated with differences in physique. In three areas the mean weight of children who shared beds was consistently lower than of those who did not.

TABLE II

Average Weight of Bed-Sharers and Non-Bed-Sharers in three different localities

Locality	*Age*	*Bed-Sharers Wt. in kilos*	*Non-Bed-Sharers Wt. in kilos*
A	6	20·3 (38)	21·0 (39)
B	6	22·0 (26)	22·9 (99)
C	14	45·5 (106)	49·8 (238)

Number of boys in brackets.

This was found to be the case even when full allowance was made for any possible effect due to difference in dietary pattern. It was also found to operate independently of social class, type of school, and number of children in the family.

The information arising from nutrition surveys over the past 18 months is, therefore—

(1) No difference in physique has been found in children with food fads or who do not take school milk or meals.

(2) Haemoglobin levels at all ages are the same as, or slightly higher, than in 1943.

(3) The proportion of boys graded as "good", "fair" and "poor" is unchanged.

Information related to the nutritional state from some other sources may be referred to:—

(1) The Reports of the Registrar General show that the maternal, infant, and neo-natal morality rates, and tuberculosis mortality rates, have fallen to new low record levels.

(2) The annual reports of those school medical officers who record the heights and weights of school children, almost without exception, show that these have increased over the past three years.

(3) Beltram and Bransby's survey of the diets of children of the lowest income groups showed satisfactory levels of intake of all nutrients.

This evidence suggests that the nutritional state of England and Wales is at present good.

A fuller report of the work which has been described here will appear at a later date.

We wish to acknowledge our indebtedness to the Medical Officers of Health of the areas concerned and to their supporting staffs, for their kindness and help in expediting this work.

References

Beltram, A. and Bransby, E. R. (1950) *This Bulletin*, **9**, 177.
Berry, W. T. C., Cowin, P. J. and Magee, H. E. (1951) *This Bulletin*, **10**, 155 & 180.
Berry, W. T. C., Cowin, P. J. and Magee, H. E., (1952) *Brit. med. J.* (in Press).
Bransby, E. R. and Hammond, W. H. (1951) *Brit. med. J.* ii, 330.
Friend, G. E. and Bransby, E. R. (1947) *Lancet* i, 677.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES

FEBRUARY, 1952

(Issued from the General Register Office, Somerset House, W.C.2)

	February 2nd	February 9th	February 16th	February 23rd	Average weekly figures for February, 1951
Scarlet Fever	1,320	1,416	1,482	1,613	875
Whooping Cough	2,655	2,686	2,550	2,648	5,405
Diphtheria	24	34	48	37	45
Measles, excluding Rubella ...	3,386	4,430	4,980	5,734	22,584
Acute Pneumonia	1,005	1,105	1,175	1,143	2,594
Meningococcal Infection ...	31	49	51	45	55
Acute Poliomyelitis (Paralytic)...	17	11	13	9	17
,, ,, (Non-paralytic)	8	6	7	5	7
Ophthalmia Neonatorum ...	30	35	27	40	33
Puerperal Pyrexia and Puerperal Sepsis	257	216	226	218	81
Dysentery...	490	592	636	591	1,073
Paratyphoid	6	6	3	6	8
Typhoid	5	2	3	3	4
Smallpox	—	—	1	18	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

British Standards Institution

Laboratory workers may be interested to learn that the British Standards Institution have recently brought out a new specification (B.S.1132: 1952) for automatic pipettes of a capacity of 2 ml. to 100 ml. The specification includes standard methods for the determination of capacity and delivery time, and tolerances for both. The sizes of glass tubing recommended for the manufacture of these pipettes are given in an appendix.

Copies of this standard may be obtained from the British Standards Institution, Sales Department, 24, Victoria Street, London, S.W.1. The price is 2s., post free.

SALMONELLA INFECTION OF HEN EGGS

by

A. Bernstein, M.B., B.S.,
Central Public Health Laboratory, Colindale.

Although there is a fairly high incidence of *Salmonella* in dried egg products and an associated human morbidity (Gibbons and Moore, 1944; Solowey, Spaulding and Goresline, 1946; Report, 1947), there appear to have been only two reported outbreaks of human salmonella infection traced to hen eggs in shell (Watt, 1945; Crow, 1946).

That a variety of organisms, including salmonellae, can penetrate the intact shell of the egg has been shown by several workers (Rettger, 1913; Scott, 1930; Brown, Combs and Wright, 1940; Haines and Moran, 1940, and others), but there is little evidence of natural infection of the egg similar to that which occurs with duck eggs. J. E. Wilson (1945) thought that he had evidence of such a natural infection with *Salm.thompson,* but later he decided that the infection was due to shell penetration from some infected duck eggs which had been kept in the same incubator (Wilson, J. E., 1948). More recently (Wilson, J. E., 1950), he has reported the isolation of *Salm. typhi-murium* from 3 successive eggs, out of a total of 30, laid by an infected hen. In this instance he was able to exclude the possibility of shell passage. Buxton and Gordon (1947), working with an infected flock, were unable to obtain evidence of yolk infection in the eggs laid. Cantor and McFarlane (1948) examined a total of 2,628 eggs, cultures being made from both shell and egg contents in 2,088, from shell only in 44 and from egg contents only in 496 instances. They isolated *Salm.pullorum* from 30 out of 2,584 (1·2 per cent.) of the contents, and other salmonellae (*montevideo* and *anatum*) from 13 out of 2,182 (0·6 per cent.) of the shells. They were not able to isolate salmonellae from shell and contents of the same egg. Carter, Powell and Borts (1950) isolated 5 strains of *Salm.pullorum* and one of *Salm.paratyphi B* from the contents of 186 samples, each consisting of 1 dozen pooled eggs. These last two surveys were made in the United States of America.

Technique

The present investigation was designed to provide information on the rate of infection with *Salmonella* of marketable eggs in England. During the period June, 1950—May, 1951 samples of eggs, as received by wholesalers and retailers, were regularly examined (Table I). Fluctuations in the numbers examined were related to a variation in available supplies.

TABLE I

Distribution of Samples by Months

Source	1950							1951					Total
	Jun.	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	
Home ...	8	5	3	2	—	1	—	—	24	18	12	18	91
Imported ...	8	11	2	2	18	15	—	5	—	—	—	—	61
Total	16	16	5	4	18	16	—	5	24	18	12	18	152

Sampling

The sampling took place at a large London depot (Ministry of Food) which receives a representative 2 per cent. of all eggs imported into the London Area. It was also possible, later, to obtain supplies of home-produced eggs from a similar depot covering southern England. The origins of the eggs, their ages, and their modes of preservation are shown in Table II. Bulk samples of 2 dozen eggs were used. These were taken at random from individual cases of 30 dozen eggs. The eggs chosen had been "candled" prior to packing so that any grossly infected eggs were excluded.

TABLE II

Distribution of Samples by Source and Age

Source	Nature and Time (in months) of Storage						Total
	Fresh	Cold		Oiled		Gas >3	
		<1	1–3	1–3	>3		
Home Produced							
Bedfordshire	18	—	—	—	—	—	18
Buckinghamshire ...	6	—	—	—	—	—	6
Cambridgeshire ...	6	—	—	—	—	—	6
Essex	18	—	—	—	—	—	18
Kent	12	—	—	—	—	—	12
Norfolk	6	—	—	—	—	—	6
Surrey	6	—	—	—	—	—	6
Ulster	10	—	—	—	—	—	10
Miscellaneous	5	4	—	—	—	—	9
Imported							
Australia—N.S.W. ...	—	—	1	1	—	—	2
,, Queensland	—	—	—	1	—	—	1
,, South ...	—	—	2	3	—	—	5
,, Western ...	—	—	—	2	—	—	2
Denmark	13	3	—	—	5	3	24
Eire	9	1	—	—	2	—	12
Gambia	—	1	—	—	—	—	1
Poland	4	2	—	—	2	—	8
South Africa	—	6	—	—	—	—	6
Total Home	87	4	—	—	—	—	91
Total Imported	26	13	3	7	9	3	61
Total	113	17	3	7	9	3	152

Note:

"Fresh" —an untreated egg up to 10 days old (usually <7 days).

"Cold" —an untreated egg kept at 0°C. and a relative humidity of 80–83 per cent.

"Oiled" —an egg dipped in a fine grade mineral oil and then kept at 0°C. and a relative humidity of 75–80 per cent.

"Gas" —an egg kept in an atmosphere of 60 per cent. CO_2 at 2·5–3·0°C.

No attempt was made to sterilize or cleanse the shells, the majority of which were free from obvious dirt, and the eggs of each batch were broken by hand into a sterile bowl. Large pieces of shell were removed with a sterile spatula, but small pieces were not disturbed. The yolks were broken and the liquid thoroughly beaten with a fresh sterile spatula to give a smooth homogeneous "mélange". Approximately 1 litre of liquid was obtained from each sample.

Media

1. Selenite-F Broth (Leifson, 1936 ; Hobbs and Allison, 1945).
2. Tetrathionate Broth (Mackie and McCartney, 1942*a*).
3. Deoxycholate-Citrate Agar (DCA) (Leifson, 1935 ; Hynes, 1942).
4. MacConkey Agar (MCA) (Mackie and McCartney, 1942*b*).
5. Wilson and Blair Agar (WBA) (Wilson, W. J., 1938).

The tetrathionate broth was freshly prepared ; the plates were poured 24 hours, at the most, before use.

Method

Five ml. amounts of the mélange were added to 20 ml. of each of the two liquid media. These were incubated at 37° C. for the requisite time and then subcultured. Two different routines of subculture were used. At first the fluid media were subcultured after 1 and 3 days on to DCA plates, which were then incubated for 24 hours at 37° C. The first 37 samples were treated in this way. After the publication of the findings of Carter, Powell and Borts (1950) this routine was changed and subcultures were made after 3 and 7 days on to DCA and WBA (MCA was included later), the plates being incubated at 37° C. for 48 hours.

This second method was tested with a batch of liquid egg that had been artificially contaminated. The organism used was a recently isolated strain of *Salm.pullorum* and it was satisfactorily recovered at both intervals of subculture.

Non-lactose-fermenting colonies on the DCA and MCA plates, and any suspicious colonies on the WBA, were picked off and examined biochemically and, if necessary, serologically. No attempt was made to classify accurately all the organisms isolated, but they were placed in broad generic groups.

TABLE III

Incidence of Main Organisms Isolated

Category	Total Samples	*Chromobacterium*		*Proteus*		"None"	
		No.	Per cent.	No.	Per cent.	No.	Per cent.
Fresh	113	100	88	14	12	11	10
Oiled	16	14	88	—	0	2	12
Other Stored ...	23	21	91	6	27	2	9
Total ...	152	135	89	20	13	15	10

Notes:

(i) "None" includes samples which yielded no organisms and samples yielding organisms other than those mentioned in the table.

(ii) The figures refer to samples containing the organisms mentioned; they are not intended to indicate the ratio of one organism to another in any particular sample, or as a whole.

(iii) Since several samples yielded more than one organism, the horizontal totals do not agree with the figures given in column 2.

Results

Altogether 152 batches (3,648 eggs), being a random sample of 54,720 eggs, were examined throughout the course of the year. During the whole of this time no member of the salmonella group was isolated.

Several other organisms were isolated from the liquid, members of the genera *Proteus* and *Chromobacterium* (*Serratia*) being most frequently found (Table III). Coliform bacilli and *Achromobacterium* were isolated less often.

It was noted that, with the exception of oil-dipped eggs, *Proteus* was isolated from stored eggs more frequently than from fresh ones.

The total number of eggs examined was small by comparison with the total egg consumption in England. Nevertheless, the fact that no *Salmonella* was isolated does suggest that the chance of human salmonella infection being caused by hen eggs, as retailed, must be small.

Summary

In the course of the examination of the contents of 3,648 eggs from various sources at home and abroad, no members of the salmonella group were isolated.

Proteus was isolated from stored eggs more frequently than from fresh ones.

Acknowledgements. My thanks are due to Dr. Joan Taylor (Salmonella Reference Laboratory) and to Dr. W. C. Cockburn (Epidemiology Unit) for their advice and help in the planning of this survey ; to Mr. G. K. Defrates and Mr. F. B. Hart (Ministry of Food, Eggs Division) and to Mr. G. L. Cooper and Mr. S. Osborne (Deputy Area Egg Officers, Ministry of Food) for arranging for an ample supply of both imported and home-produced eggs ; to Mr. E. G. Martin (Ministry of Food) for technical advice on the preservation of eggs ; and to Mr. W. B. Betts (London Branch Manager, National Egg Distributors Ltd.) for the provision of facilities for sampling.

References

Brown, E. G., Combs, G. R. and Wright, E. (1940) *J. Amer. med. Ass.*, **114,** 642.
Buxton, A. and Gordon, R. F. (1947) *J. Hyg., Camb.*, **45,** 265.
Cantor, A. and McFarlane, V. H. (1948) *Poultry Science*, **27,** 350.
Carter, M. J., Powell, M. S. and Borts, I. H. (1950) *Publ. Hlth. Rep., Wash.*, **65** 778.
Crow, M. (1946) *J. Hyg., Camb.*, **44,** 342.
Gibbons, N. E. and Moore, R. L. (1944) *Canad. J. Res., Sec. F*, **22,** 48.
Haines, R. B. and Moran, P. (1940) *J. Hyg., Camb.*, **40,** 453.
Hobbs, B. C. and Allison, V. D. (1945) *This Bulletin*, **4,** 68.
Hynes, M. (1942) *J. Path. Bact.*, **54,** 193.
Leifson, E. (1935) *J. Path. Bact.*, **40,** 581; (1936) *Amer. J. Hyg.*, **24,** 423.
Mackie, T. J., and McCartney, J. E. (1942*a*) " Handbook of Practical Bacteriology " (6th ed.), p. 439, Livingstone, Edinburgh; (1942*b*) *Ibid.*, p. 137.
Report (1947) *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 260.
Rettger, L. F. (1913) *Storrs agr. Exp. Stn., Tech. Bull.*, No. 75.
Scott, W. M. (1930) *Brit. med. J.*, ii, 56.
Solowey, M., Spaulding, E. H. and Goresline, H. (1946) *Food Res.*, **11,** 380.
Watt, J. (1945) *Publ. Hlth. Rep. Wash.*, **60,** 385.
Wilson, J. E. (1945) *Vet. Rec.*, **57,** 511; (1948) *Ibid.*, **60,** 615; (1950) *Ibid.*, **62,** 449.
Wilson, W. J. (1938) *J. Hyg., Camb.*, **38,** 507.

XYLOSE FERMENTATION BY SHIGELLA SONNEI

G. H. Tee, M.A., M.R.C.S., Public Health Laboratory, Dorchester.

Ørskov and Larsen (1925) were the first investigators to report fermentation of xylose by *Shigella sonnei*. Bojlén (1934), examining the biochemical reactions of Sonne dysentery strains isolated in Denmark from 741 patients, found that 131 (12·7 per cent.) of 1,032 strains fermented the pentose xylose. These tests were repeated with similar findings except that a few strains previously failing to ferment xylose were found to do so when re-tested. Bojlén classified his strains into four groups according to whether they fermented xylose and according to their rate of fermenting maltose. Koser, Reiter, Bortniker and Swingle (1930) tested 19 strains of which none fermented xylose. Glynn and Starkey (1939) examined 54 strains (42 isolated in Canada) and stated that xylose was "very rarely" fermented. Hammarström (1949) in a careful survey of *Sh.sonnei* in Sweden examined 1,716 strains of which 238 (13·8 per cent.) fermented xylose.

Biochemical differences between the organisms from smooth and rough colonies were reported by Ørskov and Larsen (1925), but other workers, notably Koser and Styron (1930*a*, *b*), Glynn and Starkey (1939) and Wheeler and Mickle (1945) were unable to distinguish biochemically between the different colonial forms.

In this country Smith (1924) examined 3 strains biochemically which failed to ferment xylose. Carter (1937) reported finding 2 xylose-fermenting strains (1·7 per cent.) among 120 examined, but Beck and Buckle (1939) found no xylose fermentation among 140 strains isolated in a hospital outbreak of Sonne dysentery. Cruickshank and Swyer (1940) found 18 xylose-fermenting strains out of 24 isolated during an outbreak in a nursery. They note that, of 40 strains from various sources isolated during the few years previous to 1940, only 4 fermented xylose, these being from sporadic cases. These workers drew attention to the value of this test in identifying epidemiologically related strains and in tracing the source and mode of spread of an epidemic.

The evidence is, therefore, that though xylose-fermenting strains of *Sh.sonnei* have been found not uncommonly in Denmark and Sweden, such strains have been seldom reported in this country.

During an investigation designed primarily for bacteriophage-typing (unpublished) 812 strains of *Sh.sonnei* collected from 12 regions were examined biochemically.

The regions from which the collection of strains of *Sh.sonnei* were obtained during a period of about 30 months were: Birmingham, Cambridge, Dorchester, Leicester, Luton, Manchester, Newcastle, Newport, Northallerton, Norwich, Nottingham and Oxford—places scattered widely over the country and representing regions in which Sonne dysentery was known to be occurring during that time.

Methods

A. *Media*

(1) *Sugar substrate.* To 99 ml. of 1 per cent. peptone water was added 1 gm. of the sugar (Kerfoot) and 1 ml. of Andrade's reagent. The preparation was sterilized by filtration. Autoclaving at 10 lb. for 10 minutes was found to be unsatisfactory for xylose.

(2) *Andrade's indicator reagent:*

Acid fuchsin	0·5 gm.
Normal caustic soda	4·0 ml.
Distilled water	100·0 ml.

To the acid fuchsin add the caustic soda and 5·0 ml. of distilled water. Boil. Make up volume to 100·0 ml. Add powdered animal charcoal (2 heaped teaspoonfuls to 100 ml.). Boil. Cool. Filter through paper.

Inoculation technique. 5 ml. amounts in tubes were inoculated with a wire, the cotton-wool plugs being waxed to prevent evaporation. It was found that the amount of initial inoculum made no difference to the results after incubation at 37° C. for 10 days.

B. *Identification*

All strains whether received in pure culture or isolated from faeces in the laboratory were examined as follows:

(1) *Colonial morphology* on MacConkey's agar after 24 hours' incubation at 37° C. Two colonial forms are commonly found in freshly isolated cultures upon this medium:

(*a*) Non-lactose-fermenting colony about 2·0 mm. in diameter, low dome-shape with slight umbonation, greyish-white colour by reflected light but rather brownish by transmitted light, entire edge, smooth shiny surface, soft consistency, easily emulsified.

(*b*) Non-lactose-fermenting colony often 4–5 mm. in diameter, colourless, flat, irregular edge, matt surface, soft consistency, easily emulsified.

(2) *Biochemical reactions.* A portion of a characteristic smooth colony (and from a rough colony in the first hundred instances) was inoculated with a wire into sugar substrates. Glucose and mannitol were fermented with production of acid (no gas) in less than 24 hours. Lactose and sucrose showed no change after 24 hours as a rule, but a few strains produced some acid within this time. Salicin, sorbitol and dulcitol were not fermented in 10 days. Indole was not produced after 48 hours' incubation in peptone water.

(3) *Motility.* Strains were tested for spread in Craigie's tubes. All were found to be non-motile after incubation at 37° C. for 10 days.

(4) *Agglutination* of an overnight broth culture diluted in normal saline and standardized by opacity tubes to about 250 million per ml. " Standards Laboratory " antiserum (S+R) was used and agglutination to a titre of 1/250 was obtained after incubation in a water-bath at 52° C. for 2 hours.

No strain was accepted as being *Sh.sonnei* unless it conformed to all these criteria.

Findings

Of the 812 strains, from 812 patients, a total of 44 (5·4 per cent.) were found to ferment xylose, with the production of acid but no gas on incubation at 37°C. for 10 days. Almost all positive reactions occurred within 24 hours' incubation time. These results were found to be constant when repeated after strains had been maintained on Dorset's egg medium for a period of at least 20 weeks and at most 2 years.

In addition to the pentose xylose, other substrates were used for testing biochemical properties. All strains were found to produce acid without gas in glucose, mannitol, maltose, lactose, sucrose and rhamnose.

It is noteworthy that all the 44 xylose-fermenting strains failed to ferment the triose raffinose. Of the 768 strains isolated which failed to ferment xylose only 19 also failed to ferment raffinose (= 2·5 per cent.).

Of the 129 strains tested for melibiose fermentation, 118 produced acid from this sugar. The 11 strains that failed to do so were all isolated in Cambridge, 5 of them within a few days of one another. It has not been possible to assess the value of biochemical tests with this sugar in providing evidence of strain similarity ; more strains need to be examined to determine this point.

In this series both smooth and rough colonies were tested separately for xylose fermentation by the first hundred strains of *Sh. sonnei* examined, and by the 44 strains fermenting xylose when smooth and rough organisms were mixed. No difference between the reactions of mixed smooth and rough cultures and the derived pure rough form was found.

The distribution of strains fermenting xylose is shown in Table I.

TABLE I

Distribution of Xylose-Fermenting Strains of Sh.sonnei

Source	Total number examined	Strains fermenting xylose
Oxford	5	1
Newcastle	23	1
Leicester	72	2
Cambridge	241	40

Discussion

The *Sh.sonnei* strains fermenting xylose isolated from Oxford and Newcastle represented sporadic cases. The two xylose-fermenting strains from Leicester were isolated from a mother and daughter in the same household.

TABLE II

Distribution of Xylose-Fermenting Strains isolated in the Cambridge Region, 1948–1951

Strain number	Date	Source
1	1948 April	Fulbourne
2	1948 December	Fulbourne
50	1949 March	Wisbech
60	1949 May	City
61	1949 May	City
63	1949 May	City
64	1949 May	City
77	1949 May	City
207	1949 July	Hospital
210	1949 July	Hospital
211	1949 July	Hospital
213	1949 July	Hospital
217	1949 July	City
218	1949 July	Hospital
219	1949 July	City
220	1949 July	City
221	1949 July	City
222	1949 July	City
223	1949 July	City
225	1949 July	Hospital
226	1949 July	Hospital
227	1949 July	Hospital
228	1949 July	Hospital
229	1949 July	Hospital
230	1949 July	Girton
231	1949 August	Hospital
232	1949 August	Hospital
233	1949 August	City
235	1949 August	City
236	1949 August	City
237	1949 August	City
238	1949 August	Hospital
239	1949 August	City
240	1949 August	City
322	1950 January	Hospital
323	1950 January	Hospital
621	1950 October	City
627	1950 October	City
630	1950 October	City
660	1951 February	City

In the Cambridge region out of a total of 241 strains isolated between 1948 and 1951 (from 241 patients) it was found that 40 (16·6 per cent.) fermented xylose. The distribution of these is shown in Table II. It is of particular interest to note that 26 xylose-fermenting strains were isolated during a few weeks in the summer of 1949 when Sonne dysentery became common in the city and 13 cases occurred in one of the hospitals among the nursing staff. All the strains of *Sh. sonnei* isolated during this outbreak in the city (including the hospital) fermented xylose. During this

period only one case of Sonne dysentery was reported from the country around Cambridge and the strain isolated from this case failed to ferment xylose.

In January, 1950, two further strains fermenting xylose were isolated from two more nurses with diarrhoea in the same hospital, but by this time three xylose-negative strains had been found in cases in the city.

In May, 1950, nine cases of diarrhoea occurred in the children's ward, eight patients and one nurse being affected. *Sh. sonnei*, which failed to ferment xylose, was isolated from each of these. Since it was known that cases of Sonne dysentery had been occurring a few months before among the nursing staff, it might have been thought possible for a nurse who was an old unknown carrier to have infected the children. But since all the strains isolated from the nursing staff in 1949 and in January, 1950, had been of the xylose-fermenting variety the explanation was not considered probable. This view was supported by the epidemiological evidence, since the first child to have diarrhoea was an infant (X) admitted with a " feeding difficulty." Two days later the other seven cases in the ward and the nurse got diarrhoea. At the house from which the first child (X) to develop diarrhoea had been admitted, two carriers of *Sh. sonnei* were found ; these two strains were also xylose-negative. The evidence is therefore that this xylose-negative strain had been introduced into the hospital from outside and that the subsequent seven cases among the children and that of the nurse were the result of ward cross-infection for which the affected nurse was not necessarily responsible.

In October, 1950, three xylose-fermenting strains were isolated from cases in the city, two of which occurred in the same family. In February, 1951, one further xylose-fermenting strain was found. All strains isolated subsequently in the Cambridge region had been xylose-negative: these numbered 139 (up to May, 1951). During the whole period there was no instance of both xylose-positive and xylose-negative strains being found in the same household or in the same case.

It would appear that xylose-fermenting strains of *Sh.sonnei* are uncommon in this country generally, but that they may prevail in one locality for a short time during which they are the common type to be isolated. No evidence of endemicity of this type has been found.

Study of the incidence and mode of spread of xylose-fermenting strains may well prove an important factor in elucidating the epidemiology of Sonne dysentery.

Summary

During a period of 35 months 812 strains of *Sh.sonnei* from 812 cases (including carriers) were obtained from 12 regions in England including Monmouthshire where Sonne dysentery was prevalent.

It was found that 44 strains out of 812 (5·4 per cent.) fermented xylose at 37°C. within 10 days. None of these xylose-fermenting strains fermented raffinose.

The epidemiological significance of these findings is discussed.

The advice of Dr. R. M. Fry, Director, Public Health Laboratory, Cambridge, and the help of those bacteriologists who so kindly supplied cultures, are most gratefully acknowledged.

References

Beck, A. and Buckle, D. F. (1939) *J. Hyg., Camb.*, **39**, 311.
Bojlén, K. (1934) " Dysentery in Denmark ". Copenhagen.
Carter, H. S. (1937) *J. Path. Bact.*, **45**, 447.
Cruickshank, R. and Swyer, R. (1940) *Lancet*, ii, 803.
Glynn, J. H. and Starkey, D. H. (1939) *J. Bact.*, **37**, 315.
Hammarström, E. (1949) " Phage-typing of *Shigella sonnei*." Stockholm.
Koser, S. A., Reiter, D. O., Bortniker, E. and Swingle, E. L. (1930) *J. prev. Med.*, **4**, 477.
Koser, S. A. and Styron, N. C. (1930 *a*) *J. infect. Dis.*, **47**, 443; (1930 *b*) *Ibid.*, **47**, 453.
Ørskov, F. and Larsen, A. (1925) *J. Bact.*, **10**, 473.
Smith, J. (1924) *J. Hyg., Camb.*, **23**, 94.
Wheeler, K. and Mickle, F. L. (1945) *J. Immunol.*, **51**, 257.

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.
Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The February Bulletin was issued on 15th February.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(14860) Wt. 8055—6219 3,300 3/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

APRIL **1952** **Vol. 11**

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1

VARIOLA MINOR

J. Pickford Marsden, M.D., D.P.H.

The existence of a mild form of smallpox, which " breeds true ", has been recognized at least since the 17th century. Jenner refers to it in his " Inquiry ". Variola minor (alastrim, amaas, kaffir pox) is now endemic over large territories, especially in Africa and in the northern half of South America. In England the disease was last endemic from 1923 to 1934. Between 1928 and 1934 some 13,686 cases of variola minor were treated in the London smallpox hospitals. The following remarks are based on a study of these cases, fuller details of which can be found in the Bulletin of Hygiene, 1948, **23,** 735.

Modified smallpox

The severity of an attack of smallpox measures the resultant of the two opposing forces, the patient's (relative) immunity and the virulence of the infective agent. In variola major the virulence is high: in variola minor it is low. Modification in variola major is determined by host resistance (relative immunity) and therefore its extent varies from case to case. In variola minor modification arises from weakness of the strain of virus and therefore is invariable: in other words, variola minor " breeds true ". But an individual case of variola minor may be indistinguishable, at the bedside, from an attack of variola major in a subject who is partially immune.

In an attack of smallpox, modified by whatever means, the majority of the lesions of the focal rash are, in comparison with the lesions of natural smallpox, small, superficial, and of rapid development; hurrying through their course, or aborting and assuming a granulomatous termination. In any one case these characteristics may be noted singly, but combination of several is more usual. The effect of modification on the patient's illness is to deprive the toxaemia of most of its lethal power, and to obviate, or in the worst cases to mitigate, symptoms associated with the maturation of the eruption and complications arising out of the suppurative process.

Mortality in variola minor

This is negligible. The overall mortality in 13,686 cases was 0·25 per cent. A toxic (haemorrhagic) form may occur—as in all acute infections (3 cases). An acute demyelinating encephalomyelitis is a grave complication (9 cases, 5 fatal).

In spite of the small size of many of the lesions the rash may attain confluence on the face (19 cases). In such cases, and with the severer forms of discrete eruptions, some degree of secondary fever is to be expected (5 per cent).

Incubation period

In variola major this is remarkably constant at 14 days from rash to rash. The impression that in variola minor this period is commonly extended awaits further confirmation. On this point evidence derived from hospital sources is of limited value. But it is pertinent to remark that the common tendency is to underestimate the age of a smallpox rash, and that this is particularly true of variola minor.

The primary fever (smallpox toxaemia) in variola minor

This is an essential feature of the illness. The temperature commonly reaches 100° to 104° F.; and the fever lasts, in most cases, for 3, 4, or 5 days. The most constant symptoms are headache (76 per cent), nausea or actual vomiting (48 per cent), backache (38 per cent), shivering or feverish feelings (34 per cent), and pains in limbs, chest or abdomen which may suggest a diagnosis of rheumatism, pleurisy or appendicitis. This initial illness may be severe enough to give rise to some anxiety, but the commonest pre-eruptive diagnosis is influenza. A toxaemic (prodromal) rash may occasionally be seen at this stage. This may be urticarial, erythematous, or petechial; but it is anything but a help in diagnosis.

The focal rash in variola minor

This may appear on the first day of illness; or its arrival may be delayed until the end of the first week. But in the majority of cases (60 per cent) the outcrop occurs on the 3rd or 4th day and may coincide with the end of the primary fever. The earliest lesions may be found almost anywhere on the upper half of the body: the centrifugal distribution is not necessarily apparent at this stage. The outcrop takes place as a gradual process on the body generally, but also, to some extent, on any one particular part. In addition to small differences in age, discernible for the most part only during the early stages, between individual elements in any one situation, a more lasting contrast, once the outcrop is complete, is seen between lesions on the upper and those on the lower parts of the body, especially between those on the face, often the site of the earliest lesions, and those on the legs, the last parts to be affected. The rash is not homogeneous in the sense that all the elements erupt and then evolve simultaneously; but the rash does not " crop " as in chicken pox.

As the rash of variola minor develops modification is made evident not through the absence of any stage which is normally attained by the focal smallpox lesion but rather by an accelerated progress, a hastening through the natural process of evolution. The rate of development varies within narrow limits from case to case; but the average time occupied by the lesion at its different stages is two days as a papule, one day as a vesicle, and three or at the most four, days as a pustule. Thus the eruption is usually vesicular on the third day following the appearance of the papules. The vesicular stage is short-lived: only exceptionally does it exceed twenty-four hours; thus this stage may entirely evade detection. By the fourth day of efflorescence pustulation is apparent: the rash attains its height on the fifth or sixth day; and early crusting is established by the sixth or seventh day of the life of the lesion.

At first the lesion is unobtrusive; a pin-head papule, perceptible to touch, and enlarging as it ages. An erythematous areola may be a marked feature of many papules and vesicles, and is often most prominent around the smallest lesions. In the, short, vesicular stage evidence of the relatively superficial situation of the lesion in the skin may be shown by the shape of the summit of the lesion and of its margins, and by the almost universal absence of loculation and umbilication. Individual lesions may be quite indistinguishable from those of chicken pox.

In size the lesions vary much, but generally each stage in the progress towards maturation is marked by some evidence of growth. Many eruptions are remarkable for their altogether diminutive, yet sometimes profuse, elements. More commonly, however, amidst a rash well modified will be found, especially on the hands or feet, a few large, bold, upstanding lesions, more deeply set

and over-shadowing their neighbours, with rounded outline and dome-shaped summit, like jewels in their setting, the " sentinel pustules ".

Ricketts, referring to modified smallpox, wrote " However much the lesions may be altered in character the scheme of their arrangement will not be influenced either by the susceptibility of the patient or by the strain of the disease "; and the principles of the distribution of the variolous eruption which he taught remain the sheet anchor of diagnosis. In some cases the rash may consist only of some dozen or even fewer small, superficial, rapidly evolving lesions: in others the eruption may be numerically severe: variations will be seen between individual components in respect of size, shape of summit and edges, depth of situation in the skin, and rate of development; *yet the general pattern of arrangement is the same in all.*

Speaking generally the rash affects the face and limbs more than the trunk. Especially in the child the incidence on the lower limbs may be as marked as on the arms, and with an emphasis on the extensor and prominent surfaces rather than on the flexor and sheltered areas. The parts of the trunk are very differently affected: the bulk of the rash here is found on the convexity of the upper part of the back and least on the abdomen, flanks and axillae. Withal the rash is *general* and *symmetrical.*

Variola minor and vaccination

Among 13,686 patients only 7 showed evidence of successful vaccination performed within the previous 10 years. No member of the staff of the smallpox hospitals contracted the disease.

The disease was seen in 1,083 patients successfully vaccinated after contact.

TABLE I

Vaccinated contacts

Day of successful vaccination on which focal smallpox rash appeared	*Number of patients*
2nd	15
3rd	20
4th	42
5th	57
6th	69
7th	99
8th	115
9th	216
10th	195
11th	143
12th	89
13th	17
14th	5
15th	1

Ricketts taught that in the main it would be accurate to say that a successful vaccination done in the first seven days of a smallpox incubation period would wholly prevent the attack, but that a patient might be vaccinated successfully as early as the 14th or even the 15th day before the outcrop and yet not escape the disease. In certain cases the vaccinial reaction does not begin to arise until a week or more after inoculation, and the rise of immunity is correspondingly deferred. Too high a measure of protection is sometimes expected in vaccinating contacts.

In tables II and III are shown details of 1,865 patients who were vaccinated after the onset of the disease.

TABLE II

Patients vaccinated after the onset of the disease but before the outcrop of the focal rash

Day of disease	Number of patients vaccinated	Vaccination successful		Vaccination unsuccessful	
		Number of patients	Percentage	Number of patients	Percentage
1st	59	46	78	13	22
2nd	58	32	55	26	45
3rd	41	23	56	18	44
4th	8	2	25	6	75
5th	5	1	20	4	80
6th	2	2			
7th	1	1			

TABLE III

Patients vaccinated after outcrop of the focal rash

Day of rash	Number of patients vaccinated	Vaccination successful		Vaccination unsuccessful	
		Number of patients	Percentage	Number of patients	Percentage
1st	377	129	34	248	66
2nd	347	65	19	282	81
3rd	282	21	7	261	93
4th	192	3	2	189	98
5th	144	2	1·5	142	98·5
6th	84	1	1	83	99
7th	69	Nil	Nil	69	100
8th (or after)...	196	Nil	Nil	196	100

A steep decline in the incidence of successful vaccination after the onset of illness is characteristic of smallpox; and a " take " after the first few days of the rash would provide very strong evidence against a diagnosis of smallpox.

MEMORANDUM ON VACCINATION

The Ministry of Health memorandum on vaccination, first published in 1948, is now available in a revised form. (Memo. $\frac{312}{\text{Med}}$ Revised London H.M.S.O. 1952, Price 4d. net.) The main recommendations of the original memorandum, e.g., regarding the use of the so called " multiple pressure " technique are unaltered, but the revised version includes notes on the completion of the vaccination record card used in the National Health Service and refers to the desirability whenever possible avoiding the coincidence of a primary vaccination with an inoculation against yellow fever. Local prevalence of poliomyelitis is mentioned as an indication for a temporary suspension of routine infant vaccination.

I. THE MAINTENANCE OF EFFECTIVE TREATMENT IN HIGH TEMPERATURE SHORT-TIME EQUIPMENT FOR PASTEURISING ICE-CREAM MIX

C. C. Thiel, B.Sc.(Agric.), Ph.D. and H. Burton, B.Eng.,

National Institute for Research in Dairying, Shinfield, Reading

When high-temperature short-time equipment is used for the pasteurisation of ice-cream mix, the problem of ensuring proper treatment is rather different from that which occurs when milk is treated by the same process. As there is no routine method such as the phosphatase test to prove that the product has been effectively heat-treated all pasteurising equipment for ice-cream mix, including H.T.S.T. plant, must be adequately instrumented, tested, and inspected regularly by the responsible authority to ensure its proper functioning. H.T.S.T. equipment used for ice-cream mix must be furnished with instruments for the measurement and recording of temperature similar to those used on milk plant. The methods of checking with standard thermometers, of inspecting charts and of ensuring the correct temperature for diversion are the same in both cases. At the holding temperature ice-cream mix is much more viscous than milk or water and the viscosity is likely to vary greatly with the composition of the mix. As the viscosity of the mix affects the holding time H.T.S.T. plant must, therefore, be so designed and operated that changes in the viscosity of the mix can only result in a holding time *longer* than 15 seconds.

Particles of any fluid can pass through the holder either in orderly straight lines, those on the outside flowing slowly and those towards the centre more and more quickly (streamline or laminar flow), or in a state of turbulence. When the flow is turbulent, those particles that pass near the walls progress as in streamline flow, but the majority intermingle constantly across the stream. The curve of forward velocity distribution across the stream in a tube is parabolic in laminar flow but is much flattened in turbulent flow. (See Fig. 1.) It is evident that for the same mean forward velocity the maximum forward velocity is greater for streamline flow than for turbulent flow.

The average holding time of all particles in the holding section of a plant may be calculated from the capacity of the holder and the flowrate. For example, if the volume of a holder is 5 gallons and the flowrate 600 gph or 10 gal/min the average holding time will be 5/10 minutes=30 seconds. The holding time of an H.T.S.T. pasteuriser is of course not the average time taken by all particles to flow through the holder but the time taken by the particles which pass through it most quickly. This holding time must be determined experimentally.

The average holding time of all particles and the holding time of particles passing through the holder most quickly can be usefully combined to give the holding efficiency. This is defined as the ratio of the shortest time to the average time, expressed as a percentage or more usually as a decimal. With pure streamline flow it follows mathematically from the velocity distribution that the holding efficiency is 0·5; that is, the fastest particle flows through the holder in just half the average time taken by all particles. Any dimensional irregularities in the holder such as the expansion at the entry, bends, thermometer pockets and the contraction at the exit will disturb the streamline flow to some extent. In practice, therefore, completely undisturbed streamline flow is not likely to be found in the holder of a plant treating ice-cream mix and the theoretical minimum efficiency of 0·5 is not likely to be encountered. With increasing turbulence in the holder the holding efficiency increases to a maximum of approximately 0·8, above which greater degrees of turbulence have no effect (Goodman 1949).

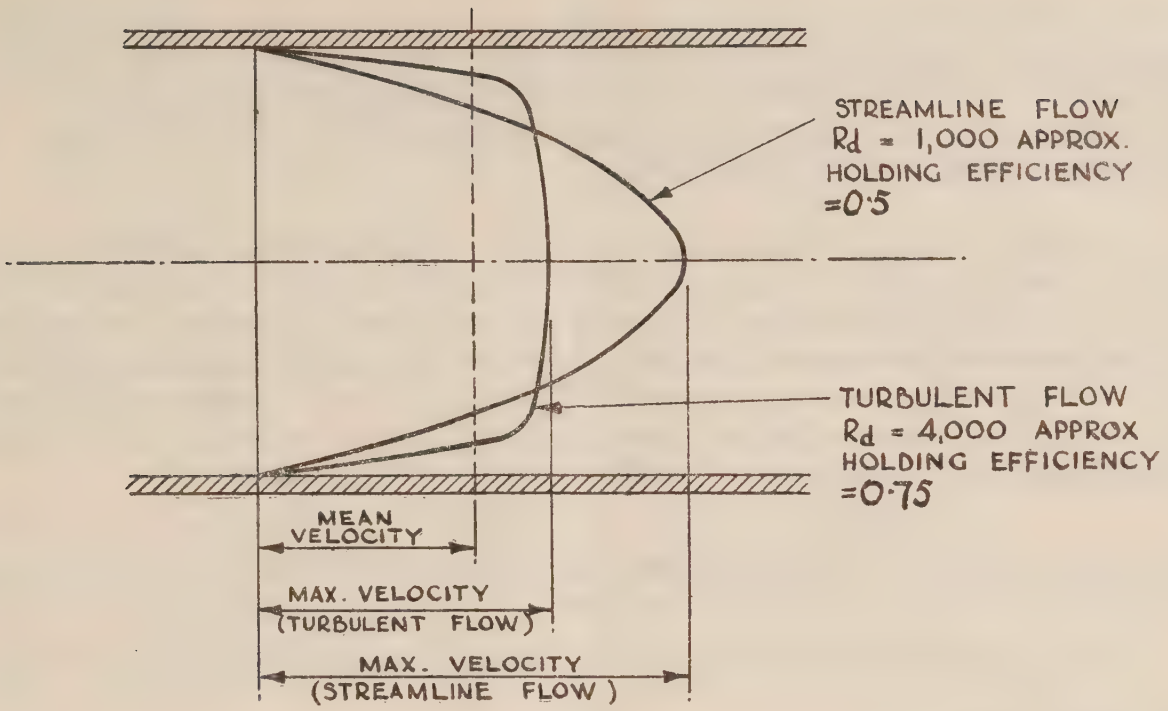


FIG. 1. Diagram of velocity distributions across a plain circular tube under conditions of streamline and turbulent flow, turbulence being induced by reducing the kinematic viscosity in the ratio 4 : 1, the mass flowrate remaining constant.

Whether flow in a straight circular tube will be laminar or turbulent is indicated by the

$$\text{Reynolds number} = \frac{(\text{average velocity}) \times (\text{diameter of pipe})}{(\text{kinematic viscosity of the fluid})}$$

For values of the Reynolds number under 2,000 the flow is streamline, from 2-3,000 it is partly laminar and partly turbulent, and above 3,000 it is turbulent. It is evident from the equation that the Reynolds number is inversely proportional to the viscosity. Therefore, if other conditions remain constant but the viscosity of ice-cream mix is allowed to vary widely it is likely that the type of flow will alter (Fig. 1). If the flow changes from turbulent to streamline the holding efficiency may change from 0·8 to 0.5. Now if the holding time of a particular plant were 15 seconds and the holding efficiency 0·8, the holding time at a holding efficiency of 0·5 and the same throughput would be $\frac{0{\cdot}5}{0{\cdot}8} \times 15 = 9{\cdot}4$ seconds. In ice-cream plant it is unlikely that the holding efficiency will ever be as high as 0·8 or as low as 0·5, but a change in holding time of several seconds following a great change in viscosity of the mix is theoretically and practically possible.

Normal ice-cream mixes are of such a kinematic viscosity that in any flow-holder of practical design the flow conditions will be streamline with a little turbulence induced by irregularities at some places along its length. It follows, therefore, that holders will normally be designed to give not less than 15 seconds holding time when flow is streamline or nearly so, and the holding efficiency will lie in the range 0·5 to 0·6. In the event of such a plant being used to treat mixes of very much lower viscosity than usual, or if it were to be tested with water instead of mix, it is likely that the conditions of flow would become turbulent giving a higher holding efficiency and a holding time greater than 15 seconds. Thus, if a plant is shown to exhibit streamline flow in the holder when mix is being treated and at the same time the holding time is shown to

be not less than 15 seconds, any subsequent alteration in mix composition cannot cause the holding time to fall below 15 seconds. Mixes of greater viscosities will not appreciably affect holding efficiency whilst mixes of much lower viscosities may increase the holding efficiency so that the holding time will become greater than 15 seconds.

One further point must be borne in mind. Holding time is directly related to the flowrate and it is essential that this rate should be constant. Plant for the H.T.S.T. processing of ice-cream mix must therefore be equipped with a positive displacement pump. A piston type of homogeniser governing the flowrate fulfils this requirement. Because of wear positive displacement pumps generally give a reduced pumping rate after a period of use: between periodic checks the flowrate is therefore not likely to increase.

Examination of H.T.S.T. pasteurising equipment designed for treating ice-cream mix.

It is suggested the following system of initial and routine testing be adopted.

A. Initial Tests

1. *Temperature*

(*a*) Using a thermometer calibrated by the National Physical Laboratory check the hot-mix temperature-recorder and indicating thermometer.

(*b*) Check the diversion temperature and the temperature at which forward flow is re-established.

2. *Holding Time*

(*a*) Measure the volume of the holder.

In most cases this can be done by plugging the holder at its lowest point and filling it with water, taking care that all air is eliminated. The water should then be collected and weighed.

(*b*) Measure the flowrate. This must be done when the complete equipment is operating on mix. Calculate the throughput in gallons per hour after noting the time to treat the mix from a previously calibrated tank or the time to fill a calibrated tank with pasteurised mix. Alternatively note the time to fill a 10-gallon can to the graduation mark with mix from the exit of the final cooling section if incorporated in the pasteurising unit.

(*c*) Measure the holding time when mix is being pasteurised. If a tracer chemical is injected at the entry of the holder and samples collected from the exit at 1 second intervals the holding time is indicated by the last tube in which no tracer can be detected. The injection apparatus of Dummett and Mongar (1944) may be employed, using nitrite as the tracer (Goodman, 1949), its presence in the samples being detected by the Griess-Ilosvay reagent (Thresh, Beale & Suckling, 1949). Full details of the method are given on page 82 of this Bulletin.

(*d*) Calculate the holding efficiency. Provided this is between 0·5 and 0·6, indicating conditions approximating to streamline flow, and the holding time is not less than 15 seconds no subsequent alteration of mix composition is likely to result in a holding time of less than 15 seconds. The following example shows the method of calculating holding efficiency.

Example: Holding time determined experimentally 15 seconds.

Capacity of the holder 3·91 gallons.

Flowrate 0·143 gallons/second (515 gallons/hour).

Average holding time $\frac{3 \cdot 91}{0 \cdot 143} = 27 \cdot 3$ seconds.

$\therefore$ Holding efficiency $= \frac{15}{27 \cdot 3} = 0 \cdot 55$.

B. Routine Tests

1. *Temperature*

   (*a*) Using an N.P.L. certified thermometer check the hot-mix recorder and the hot-mix indicating thermometer.

   (*b*) Check the diversion temperature and the temperature at which forward flow is re-established.

   (*c*) Inspect the recorder charts.

2. *Holding Time*

   (*a*) Inspect the plant to ascertain that no alterations have been made.

   (*b*) Measure the flowrate. This should not be greater than the figure recorded at the initial tests.

References

Dummett, G. A. & Mongar, J. L. (1944), Dairy Industries, **9**, 4, 264.

Goodman, H. F. (1949), 12th Int. Dairy Congress Proceedings, **3**, 57.

Thresh, Beale & Suckling (1949), " The Examination of Waters and Water Supplies ", J. and A. Churchill, London.

II. DETERMINATION OF THE HOLDING TIME OF PLANTS PASTEURISING ICE CREAM MIX

G. H. Botham, B.Sc., F.R.I.C. (A.P.V. Co. Ltd.)

Of the methods in common use for determining the holding time of H.T.S.T. equipment, the most familiar in this country is that described by Dummett and Mongar([1]). The general question of flow holding in pasteurisation plants has been discussed by Goodman([2]).

The Dummett and Mongar test depends essentially upon the detection of traces of nickel ions by dimethylglyoxime, but its use is limited to liquids not intended for subsequent human consumption. While it has been the practice to assume that the holding time found by this method, using water, applies also to milk and similar liquids, difficulty is always introduced in making such assumptions, for holding times are subject to variation depending on the Reynolds number as well as the flow rate. Therefore it would be desirable to find a method whereby holding times could be carried out on the actual liquid the plant is pasteurising.

In view of the popularity of the Dummett and Mongar method for water, it was felt that the most convenient approach would be to find a chemical which could be injected into the product stream and be easily detectable at very low concentrations, and using essentially the same technique as for water. The chemical chosen must obviously be non-toxic, water-soluble and reasonably stable. Detection would preferably be by the formation of a pink coloration, as in the case of the nickel dimethylglyoxime reaction.

In the case of milk, most of these requirements were found to be satisfied by sodium nitrite, which can be detected in extremely dilute solutions by the well-known Griess-Ilosvay test, which gives a red colour. Laboratory work showed that, in milk warmed to pasteurising temperature, the test gives a recognisable pink colour in the presence of as little as one part of added sodium nitrite in two million parts of milk, and some observers claim to detect down to one part in ten million parts of milk.

Holding time tests done in a commercial pasteuriser, deliberately made inefficient to exaggerate any difference between the nickel chloride and the sodium nitrite tests, gave the following results for holding efficiency:—

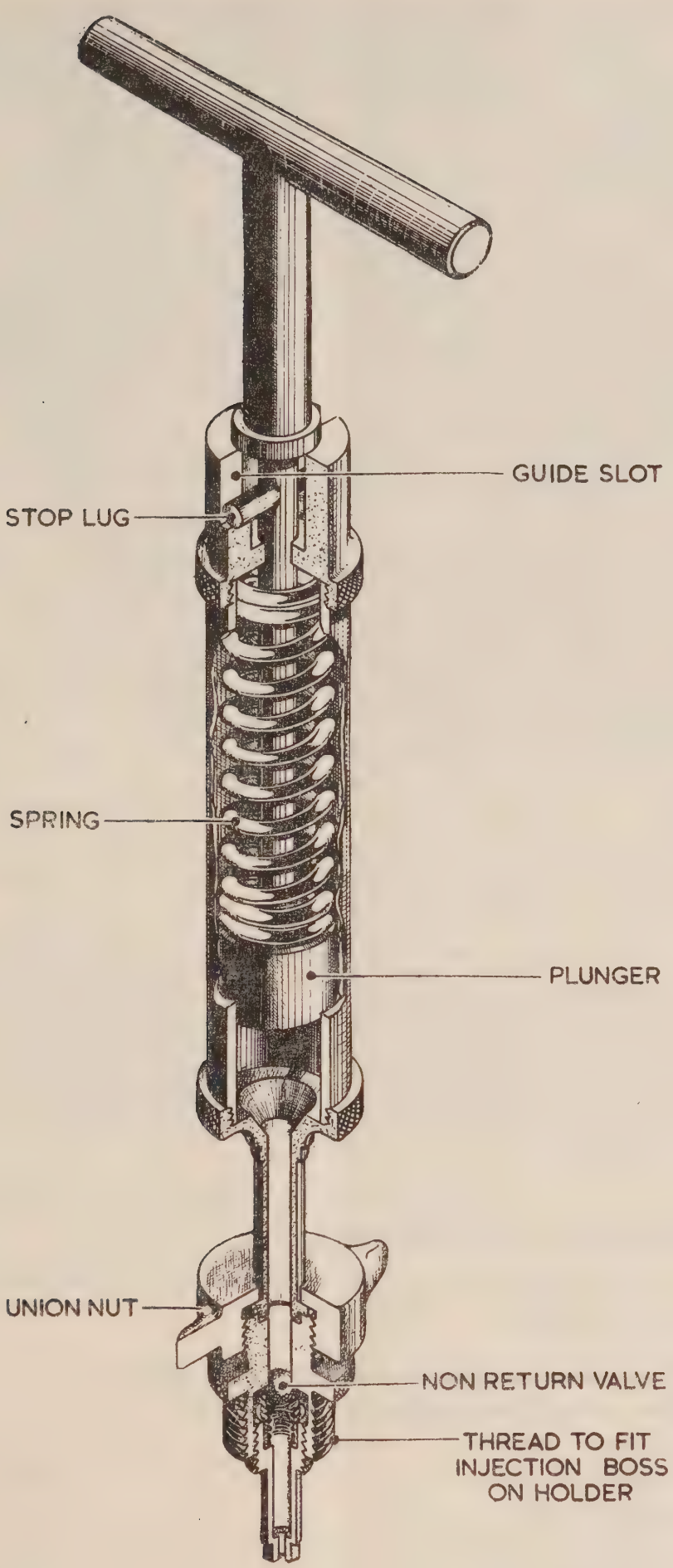
	Nickel Chloride (six tests) Per cent.	*Sodium Nitrite (six tests) Per cent.*
Maximum	60	59
Minimum	57	55½
Mean	58½	57

(A difference of 1 per cent. efficiency represents 0·2 sec. holding time.)

It would seem, therefore, that on water the sodium nitrite method is slightly more sensitive than the nickel chloride in turbulent flow conditions.

Another series of tests was run on the latest design of holding tube, using water and milk successively, and it was shown that, while the nickel chloride test gave 17 secs., the sodium nitrite test gave a holding time of 16½ secs. In the case of the sodium nitrite test on milk at pasteurisation temperature, the colour formation is practically instantaneous and the white background, provided by casein precipitated by the Griess-Ilosvay reagent, greatly enhances the ease of detection of the first faint pink colour.

It is certain, therefore, that the holding time for a particular piece of apparatus in turbulent flow conditions is given with sufficient accuracy by both nickel chloride/dimethylglyoxime on water, or sodium nitrite and Griess-Ilosvay on milk.



INJECTION GUN

(Fig. 2)

However, in liquids such as ice cream mixes at pasteurising temperature, with viscosities markedly higher than that of water, it may happen that flow through the holder is within the transition range between laminar and turbulent flow, or it may even be laminar, in which conditions particles in midstream may travel markedly faster than the average. Where these conditions exist, it is no longer valid to assume that the holding time of the ice cream mix at a given rate of flow is the same as that of water at the same flow rate as given by the nickel method. Thus, where it becomes necessary to check the holding time of apparatus used for pasteurising ice cream mixes, the test is done preferably under the actual operating conditions. It is here that the nitrite test has been particularly serviceable in giving an objective measure of the minimum time of holding of any particle of the ice cream mix, and a full description of the method employed in carrying out this test is given below:—

The special apparatus required is fully described by Dummett and Mongar (*loc. cit.*). It consists of a gun for injecting sodium nitrite solution, a suitable injection nozzle and adaptor to screw into the holder inlet, a pipette for filling the gun, a sampling valve at the holder outlet and a set of test tubes of special design.

Details of the gun and nozzle are shown in Fig. 2. The gun consists of a spring-loaded syringe with a simple release device at the top. To load, the plunger is pulled up, pressing the spring until the stop-lugs are above the guide slots in the cap. A slight twist of the handle keeps the plunger in this position. After the gun has been filled, it is screwed on to the nozzle by means of the union nut and the plunger is released by a further twist of the handle. The spring exerts a maximum pressure of 100 lbs./sq. in. on the plunger, which is sufficient to ensure quick injection against the high back pressure likely to be encountered on any pasteurising plant. This is essential, if the test is to be precise.

The nozzle and its adaptor are screwed into a boss suitably placed at the inlet of the holder, so that the liquid is sprayed out at right angles to the main direction of the flow through the plant. This gives minimum disturbance of the flow and produces the short wave front which is necessary for accurate timing. The nozzle contains a non-return valve to prevent the nitrite solution diffusing into the holder before injection takes place. The charged gun is screwed into the adaptor just before the test is carried out.

The sampling apparatus is shown in use in Fig. 3.

The sampling valve is adjusted so that 70 mls. per sec. are flowing out through the sampling valve into the test tubes. These test tubes have square tops and are held in a rack with the tops touching, so that the stream can be sampled without loss. Each tube, marked at 70 mls. is filled in succession so that the time taken to fill one tube is one second. Time in seconds from the start of sampling is thus automatically measured by the number of tubes filled. The Griess-Ilosvay reagent is measured into the tubes before sampling. Any colour in a filled tube therefore indicates the presence of nitrite solution which has passed through the holder in a time corresponding to the tube in question.

Procedure

1. Screw the adaptor/injection nozzle and the sampling valve into suitable fittings at the inlet and outlet of the holder respectively.

2. Open the sampling cock and adjust to deliver 70 mls. per second as measured in a simple measuring cylinder. (The indicator tube mentioned by Dummett and Mongar (1944) is not now used.)

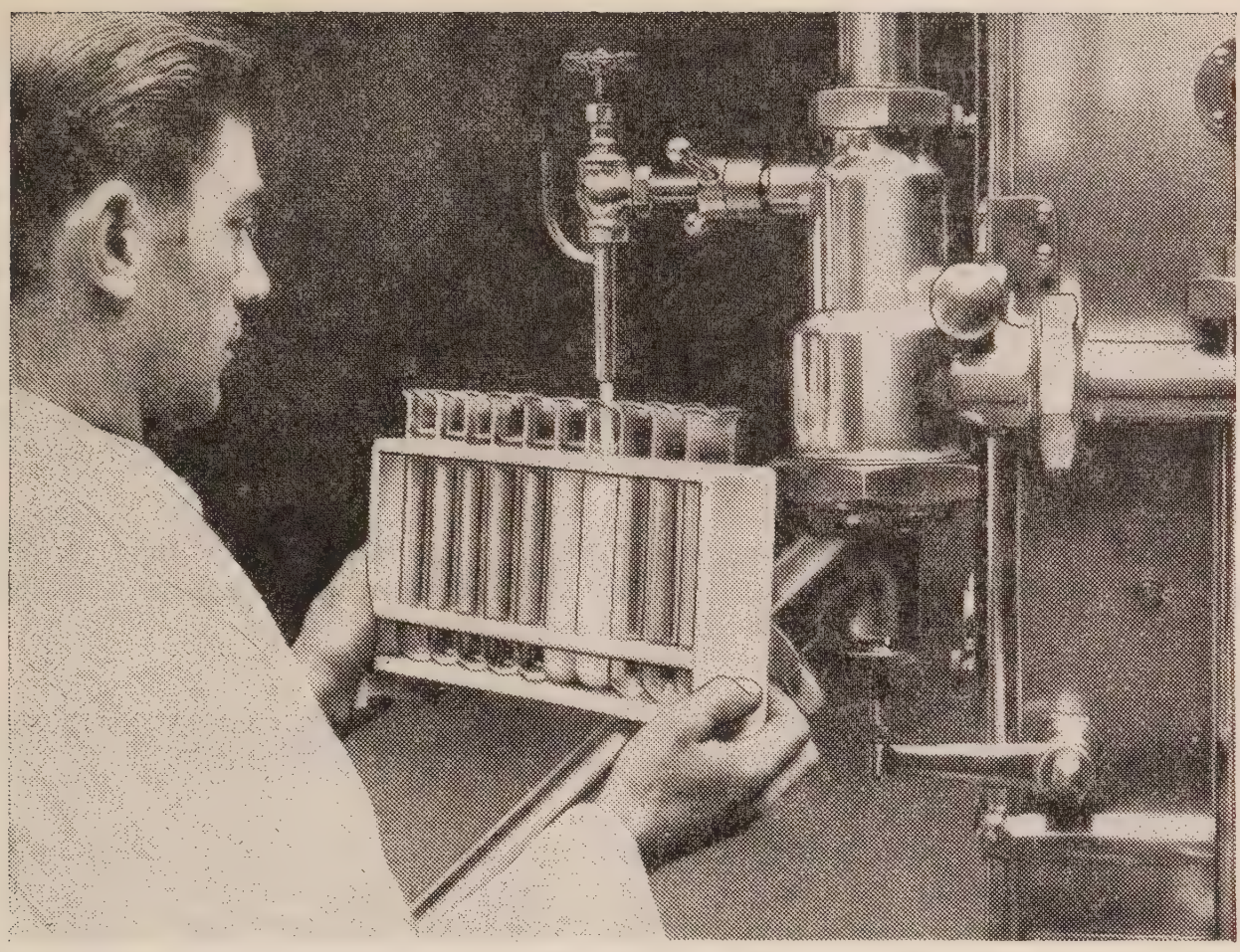
3. Add 5 mls. of the Griess-Ilosvay reagent to each test tube.

   This solution is made up as follows:—

(1)	Sulphanilic acid	0·5 gm.
	Glacial acetic acid	30 ccs.
	Distilled water	120 ccs.
(2)	Naphthylamine (alpha)	0·1 gm.
	Glacial acetic acid	30 ccs.
	Distilled water	120 ccs.

   Dissolve the naphthylamine in water at boiling-point. Cool and add acetic acid and filter.

   Mix the two solutions. Keep in a stoppered bottle.



(Fig. 3)

4. Load the gun and fill with a 10 per cent. w/v solution of sodium nitrite in distilled water. Inject by turning the handle of the gun slightly to release the plunger. At the same instant start the stop watch.
5. As the stop watch reaches 10 seconds, pass the sampling rack under the stream in such a way that the sampling tube is changed quickly at each second interval. Observe the minimum number of colourless tubes; this number plus 10 gives the holding time in seconds. If none of the tubes is coloured, repeat the test, starting to sample at 15 to 20 seconds. The average of three tests should be taken; individual results should not differ by more than one second. The plant should be run for ten minutes after each test to ensure proper clearance of the sodium nitrite from the system.
6. The tubes must be carefully cleaned after each test and reasonable precautions taken to avoid contamination of the sampling tubes with the sodium nitrite solution accidentally transferred by the hands of the operator.

Note: It is obvious that this test would not be valid if any of the ingredients of the product being used contained nitrite originally.

References:

1. Dummett, G. A. and Mongar, J. L. (1944) Dairy Industries, **9**, 4, 264.
2. Goodman, H. F., 12th Int. Dairy Congress Proceedings, **3**, 57.

SURVEY OF SICKNESS—SEPTEMBER QUARTER 1951

(Issued from the General Register Office, Somerset House, W.C.2.)

The Registrar General's Quarterly Return No. 412 (in Tables A to H) gives detailed results of the Social Survey's interviews of random samples of the adult population carried out in August, September and November, 1951, in which the experience of July to September was recorded. The report given here presents some rates derived from these tables without standardisation or correction for the number of days in the month and compares them with those for preceding months. More detailed studies appear from time to time in the publications of the General Register Office.

Owing to a General Election in October, no interviews were held in that month, but the rates shown for August and September have been adjusted to allow for the absence of these interviews.

Since the beginning of 1951 results from the Survey of Sickness have related only to persons aged 21 and over, and not 16 and over as formerly.

The " Monthly Health Index "—the proportion of persons per 100 interviewed who suffered no illness or injury, so far as they remembered, during the stated month—is given in Table A, separately for men and women, at ages 21–64 and ages 65 and over. The index was slightly lower for men under 65 and for men and women 65 and over during September, 1951, than for the corresponding month in 1949 and 1950.

Table B gives monthly illness rates, the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only, distinguishing persons with illness commencing in the month from those with illness continued from the previous month. The table also gives days of incapacity reported in each month and numbers of medical consultations. Including persons with an injury the average rates for the September Quarters of 1949, 1950 and 1951 compare thus:—

	Ages 21–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
July–Sept., 1949 ...	63	75	40	81	95	59
July–Sept., 1950 ...	65	70	39	84	100	67
July–Sept., 1951 ...	65	67	38	82	77	60
July–Sept., 1951 per cent. of July–Sept.,						
1949	103	89	95	101	81	102
1950	100	96	97	98	77	90

The level of sickness during these three September Quarters remained practically unchanged. Days of incapacity and numbers of medical consultations at ages 21–64 continued to decrease compared with July—September,

1949. At ages 65 and over incapacity was considerably less than in 1949 and 1950, while the medical consultation rate fell to approximately the same level as in 1949.

Table B also shows that during the months July to September there was the usual slight reduction in rates compared with the June Quarter.

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during each month from those who developed an illness of a minor or ill defined nature. Average monthly rates during the September Quarters of 1949, 1950 and 1951 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	Ages 21–64		65 and over		Ages 21–64		65 and over	
	M	F	M	F	M	F	M	F
July–Sept., 1949	2·9	3·9	3·6	5·2	26·4	34·3	28·5	30·0
July–Sept., 1950	3·6	4·5	5·4	6·4	31·4	39·3	28·1	36·5
July–Sept., 1951	4·5	5·2	5·1	9·1	30·4	38·6	29·5	35·5

The rates of more serious illnesses were slightly higher in the September Quarter of 1951 than in the September Quarters of the two previous years, but the rates of minor illnesses though higher than in 1949 were little different from the corresponding months of 1950.

Table D gives average monthly numbers of new illnesses from three selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the number of days of incapacity arising from each cause (including both new and continued illness). In this table the rates for the younger group relate to ages 16–64 in 1949–50 and to ages 21–64 in 1951 so that the rates shown for 1949 and 1950 would need to be increased by a small fraction to be properly comparable with those for 1951 (see this Bulletin, October, 1951, page 241).

The incidence of influenza and colds continued to decline from the high level experienced during the epidemic at the beginning of 1951 and fewer cases were reported during July to September, 1951 than in the corresponding quarter of the two previous years. Incapacity attributed to these causes was also low compared with the two previous September Quarters.

On the other hand the incidence of other respiratory diseases tended to be higher than in the September Quarters of 1949 and 1950. The incidence of rheumatism was also higher than in 1949 and 1950; but the amount of incapacity attributed to rheumatism while higher for the 21–64 group, was appreciably lower for ages 65 and over than during July to Septem ber, 194 and 1950.

Days of incapacity from all causes per 100 persons interviewed by sex and age are shown below.

	Ages 21–64		Ages 65 and over	
	Males	Females	Males	Females
1949				
January–March	122	127	200	220
April–June	86	75	151	165
July–September	82	70	89	99
October–December	98	99	225	302
1950				
January–March	114	117	196	231
April–June	89	76	143	133
July–September	79	62	118	87
October–December	99	96	182	176
1951				
January–March	167	177	295	271
April–June	86	68	126	112
*July–September	80	58	88	73

* Corrected for absence of October interviews.

The average monthly incapacity rates in the September Quarter, 1951 were in general lower than in the two preceding years. The rates during this quarter were also much lower than in the June Quarter, 1951.

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 21–64						Ages 65 and over					
	M			F			M			F		
	1949	1950	1951	1949	1950	1951	1949	1950	1951	1949	1950	1951
January	35	33	28	25	25	21	20	21	15	12	12	8
February	34	33	31	23	26	24	18	20	16	9	11	10
March	34	37	33	23	27	26	16	20	14	12	11	12
April	39	37	36	26	29	29	19	18	20	12	11	12
May	39	41	40	28	28	30	23	21	23	15	12	12
June	42	42	41	30	30	32	24	21	23	14	12	13
July	43	40	41	31	31	31	26	22	21	14	12	15
August	43	42	41*	31	31	31*	26	25	24*	16	11	16*
September ...	44	39	35*	31	29	29*	25	20	18*	15	13	11*
October	39	36		26	25		24	22		14	11	
November ...	35	37		26	27		23	22		13	11	
December ...	36	34		27	26		27	20		14	12	

(Based on combined results of interviews in two following months.)

* Experience based on one month's interviews corrected by factors based on previous experiences.

B.—*Monthly Illness Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 21–64				Ages 65 and over			
	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month
1949								
January ...	44·4	25·2	112	48	44·2	39·7	217	72
February ...	46·9	24·7	132	51	44·7	41·3	207	71
March ...	45·9	25·7	131	47	43·7	42·1	211	83
April	39·7	27·9	87	41	38·5	46·5	175	74
May	38·3	28·0	76	41	33·1	48·1	163	61
June	34·5	28·9	78	39	32·6	47·8	142	60
July	33·5	29·4	79	42	33·1	46·6	118	63
August ...	32·8	29·5	72	38	32·7	46·9	71	54
September ...	36·1	26·1	75	39	35·9	44·6	96	60
October ...	44·0	23·3	94	43	40·6	40·9	212	59
November ...	47·0	21·7	105	42	42·8	39·4	293	73
December ...	50·9	20·5	102	42	46·3	36·5	323	65
1950								
January ...	43·9	23·2	115	43	42·2	39·7	212	68
February ...	47·1	22·9	119	51	43·9	40·3	211	77
March ...	44·8	22·9	111	50	42·2	43·3	221	87
April	41·7	24·7	87	42	42·6	43·5	183	85
May	41·2	24·2	80	43	40·5	43·9	133	80
June	37·6	25·9	80	43	36·9	47·0	94	64
July	38·2	25·4	71	38	39·5	44·1	76	65
August ...	38·4	24·3	64	37	37·5	44·9	104	62
September ...	42·5	23·3	75	41	39·8	44·1	120	73
October ...	47·6	21·7	85	44	44·8	39·5	137	61
November ...	47·3	20·6	90	43	45·5	38·4	170	70
December ...	50·4	19·4	117	42	48·0	35·7	227	73
1951								
January ...	56·5	18·5	233	60	54·9	33·7	371	88
February ...	49·7	22·0	166	54	47·4	39·5	271	87
March ...	47·9	22·6	118	49	45·7	41·5	202	80
April	43·0	24·1	85	45	41·1	43·2	153	74
May	40·7	24·1	79	42	42·3	40·6	112	70
June	40·1	23·0	66	39	42·4	40·2	87	65
July	39·9	23·4	67	38	41·2	41·0	75	63
August* ...	41·7	24·2	64	37	42·3	38·6	64	53
September* ...	37·4	25·9	71	40	38·3	44·4	96	63
October ...								
November ...								
December ...								

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury see Registrar General's Quarterly Returns, Table E.)

* Uncorrected rates based on one month's experience.

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 21–64		Ages 65 and over		Ages 21–64		Ages 65 and over	
	M	F	M	F	M	F	M	F
1949								
January ...	5·2	5·7	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	8·1	6·8	10·9	37·3	41·5	31·6	38·5
March ...	5·8	7·8	7·9	11·7	35·5	41·8	34·3	33·0
April	3·6	4·7	6·2	6·4	30·1	40·0	30·3	33·4
May	3·4	3·8	2·9	3·9	29·5	39·1	28·7	30·3
June	2·6	3·6	3·0	6·3	26·2	35·7	24·9	29·9
July	2·7	3·8	3·7	6·9	25·6	33·9	26·2	28·5
August ...	2·7	3·6	4·4	4·0	25·4	33·1	27·8	29·1
September ...	3·2	4·2	2·7	4·8	28·1	35·8	31·5	32·3
October ...	3·6	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·8	5·4	8·5	9·7	38·1	45·0	32·7	34·1
December ...	4·5	6·8	5·4	9·7	42·3	47·7	32·4	42·2
1950								
January ...	3·7	5·8	9·6	8·0	35·5	42·0	28·0	37·2
February ...	6·0	8·0	9·2	9·2	36·5	43·0	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·6	42·0	29·4	33·8
April	5·4	5·6	5·5	10·6	33·3	38·8	33·7	34·4
May	4·6	4·9	5·5	6·6	31·8	40·4	29·8	37·8
June	3·2	4·1	3·6	5·7	28·3	38·8	27·9	34·9
July	3·3	4·5	6·1	6·6	30·5	37·6	27·0	37·6
August ...	3·2	4·3	4·6	5·7	29·7	38·9	26·2	36·9
September ...	4·3	4·8	5·5	7·0	34·1	41·3	31·0	35·0
October ...	5·5	7·0	8·6	10·0	38·3	43·8	32·5	37·1
November ...	6·6	7·6	9·7	10·7	36·0	43·8	32·9	36·7
December ...	9·5	10·9	12·1	17·0	37·0	42·8	29·8	35·8
1951								
January ...	15·6	19·5	18·2	27·2	38·0	39·6	31·2	31·9
February ...	10·4	12·5	13·5	16·2	35·2	40·8	31·6	33·0
March ...	7·1	8·8	11·3	13·2	36·9	42·4	32·3	34·0
April	4·8	6·1	8·4	10·9	34·0	40·6	29·7	32·3
May	4·6	5·4	6·5	8·9	30·9	39·9	33·8	34·9
June	3·9	4·6	5·0	8·5	32·0	39·0	34·4	36·2
July	3·7	4·7	5·7	7·3	31·4	39·4	33·5	35·1
August* ...	4·3	4·1	5·0	8·0	32·6	41·9	31·7	38·0
September* ...	5·5	6·7	4·5	12·1	27·2	34·6	23·4	33·3
October ...								
November ...								
December ...								

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. "Ill-defined" excludes all symptomatic illness which caused incapacity for Work, such cases being classed to the appropriate higher category. "Illnesses starting in each month" include new and recurrent illnesses.

* Uncorrected rates based on one month's experience.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 21–64*		65 and over		Ages 21–64*		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1949								
January–March ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October–December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...	19·3	19·5	13·9	13·7	15·7	22·3	36·8	31·6
1951								
January–March ...	22·9	22·5	17·2	18·0	67·3	81·4	78·3	103·2
April–June	7·6	6·5	6·2	4·6	5·6	5·3	9·4	5·9
July–September ...	5·4	4·9	3·1	2·7	2·4	3·4	0·2	4·3
October–December ...								
	Other respiratory disease							
1949								
January–March ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...	5·6	6·6	7·4	7·1	15·5	15·5	35·1	48·6
1951								
January–March ...	6·4	6·7	8·5	7·9	25·3	30·2	83·5	73·5
April–June	4·9	4·7	6·3	6·2	14·0	6·4	25·8	20·5
July–September ...	4·6	4·3	5·3	6·1	8·6	4·5	9·7	8·5
October–December ...								
	Rheumatism, all forms							
1949								
January–March ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	9·2	22·2
July–September ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...	6·5	9·1	9·3	13·1	4·7	5·8	6·3	16·9
1951								
January–March ...	8·2	10·5	10·4	14·5	10·9	7·3	18·6	18·0
April–June	6·3	9·1	11·3	15·0	4·8	5·2	8·5	11·0
July–September ...	6·3	8·6	8·5	14·0	6·8	6·2	7·7	5·4
October–December ...								

Note.—The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e. new, recurrent, or continued from the previous month).

* Rates for 1949 and 1950 include persons aged 16–20.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, MARCH, 1952

(Issued from the General Register Office, Somerset House, W.C.2.)

	Mar. 1	Mar. 8	Mar. 15	Mar. 22	Mar. 29	Average weekly figures for Mar., 1951
Scarlet Fever	1,506	1,581	1,547	1,486	1,403	973
Whooping Cough	2,792	2,918	3,091	3,133	3,110	5,089
Diphtheria	33	38	53	33	29	47
Measles, excluding Rubella ...	6,234	6,884	7,122	6,885	6,953	29,857
Acute Pneumonia	1,061	1,143	953	848	801	1,045
Meningococcal Infection ...	35	57	46	48	38	46
Acute Poliomyelitis (Paralytic) ...	15	13	13	12	10	15
,, ,, (Non-Paralytic)	4	6	9	8	2	4
Ophthalmia Neonatorum ...	31	46	33	54	32	40
Puerperal Pyrexia and Puerperal Sepsis	274	232	240	266	268	72
Dysentery	647	639	642	516	592	1,348
Paratyphoid	12	6	7	6	12	8
Typhoid	2	—	4	1	1	6
Smallpox	7	62	6	16	18	—

No cases of Cholera or Plague.

1 case of Typhus Fever (not louse borne) Imported. Week ended March 15th.

VENEREAL DISEASES

Analysis of the total number of new patients attending the clinics in England and Wales during the quarter ending 31st December, 1951.*

Number of patients* attending for the first time during the three months ending 31st December, 1951, and diagnosed as follows:	M	F	M	F	M	F	Totals
(*a*) Syphilis, primary	197	35					
(*b*) Syphilis, secondary	77	74	329	167			
(*c*) Syphilis, latent in 1st year of infection	55	58					
(*d*) Syphilis, cardio-vascular† ...	105	50					
(*e*) Syphilis, of the nervous system†	151	97	670	618	1,105	920	2,025
(*f*) Syphilis, all other late or latent stages†	414	471					
(*g*) Syphilis, congenital (under 1 year)	18	9	106	135			
(*h*) Syphilis, congenital (over 1 year)	88	126					
(*i*) Gonorrhoea					3,847	810	4,657
(*j*) Chancroid					125	3	128
(*k*) Lymphogranuloma inguinale					16	1	17
(*l*) Granuloma venereum ...					—	—	—
(*m*) Non-gonococceal urethritis (males only)					2,647	—	2,647
(*n*) Any other conditions requiring treatment					2,748	2,058	4,806
(*o*) Conditions not requiring treatment					6,557	3,067	9,624
(*p*) Conditions still remaining undiagnosed					245	150	395
					17,290	7,009	24,299

* Patients who have previously received treatment for the same condition at any Treatment Centre, or by a General Practitioner approved under Ministry of Health Circular 2226 are excluded.

† In order to avoid duplication, patients with cardio-vascular syphilis who are also suffering from syphilis of the nervous and/or other systems are recorded as suffering from cardio-vascular syphilis alone.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Cardiff Laboratory: Change of Telephone Number

The number Cardiff 8288 has now been replaced by a new number, Cardiff 29110.

Middlesbrough Laboratory: Change of Telephone Number

The number of this laboratory is now Middlesbrough 87766.

A PROGRESS NOTE ON FIELD RESEARCH ON POLIOMYELITIS

In February, 1951, a letter signed by Sir Harold Himsworth, Secretary of the Medical Research Council, and by Dr. J. M. Gibson, then President of the Society of Medical Officers of Health, was sent to all medical officers of health asking in general terms for their collaboration in a series of investigations on poliomyelitis. Since then preliminary inquiries have been made along four separate lines.

1. *Cases of poliomyelitis occurring after recent prophylactic inoculation or tonsillectomy*

Medical officers of health have been sending to the Medical Research Council a completed report card on every case notified from the beginning of the second quarter of 1951. The report cards give the age and sex of the patients and state whether there was a history of prophylactic inoculation or tonsillectomy within 12 months before the onset of poliomyelitis.

So far 100 cases in which there was a history of prophylactic inoculation or tonsillectomy within three months before the onset of poliomyelitis have been carefully investigated by the medical officer of health and the clinician concerned, in collaboration with a special investigator. It would be misleading if results from such a small number of cases in an inter-epidemic year were to be given at this stage, but the method adopted is giving reliable information on the apparent association between recent prophylactic inoculation or tonsillectomy and poliomyelitis.

All medical officers of health therefore are requested to continue to provide the Medical Research Council with the information about previous prophylactic inoculation and tonsillectomy in cases of poliomyelitis as requested on the special yellow report cards. Further supplies of the cards can be obtained from the Medical Research Council, 38 Old Queen Street, London, S.W.1.

2. *Records of numbers of inoculations given in welfare and school clinics*

In order to estimate the *risk* of poliomyelitis from different antigens in persons of different ages, medical officers of health of County Boroughs, irrespective of size, and of urban areas with a population of more than 100,000 persons (including London and Middlesex County Councils) have been sending weekly to the Medical Research Council records of all injections given in their clinics. The figures obtained during 1951 have not been analysed in detail, as the number of cases of poliomyelitis during 1951 was inadequate for a true assessment of the risk. It is hoped that medical officers of health who are already sending returns of the numbers of inoculations given will continue to do so during 1952. Further supplies of the necessary forms can be obtained from the Medical Research Council, 38 Old Queen Street, London, S.W.1.

3. *Inquiry into the role of "activating agents" in poliomyelitis*

In order to obtain information on the role of trauma, operations, inoculations other than prophylactic inoculations, illnesses, and physical activity as predisposing factors in poliomyelitis a controlled inquiry was instituted on a small scale in Cardiff, Devonshire, East Anglia, Exeter, Leeds, parts of London, Manchester and Oxford. Experience has shown the need for modification in the plan of this inquiry, which is now being reconsidered.

4. *Virological studies of communities*

Examination of sewage by the sewer-swab technique was carried out in collaboration with medical officers of health in 96 small urban communities, each of which was examined twice during 1951. The investigation of the swabs for the presence of virus has not yet been completed but valuable information is being obtained and it is proposed to continue this study in a smaller number of communities over a period of years.

G. S. Wilson,

Chairman,

Medical Research Council Committee on Inoculation Procedures and Neurological Lesions

ISOLATION OF A STRAIN OF MUMPS VIRUS

By E. T. C. Spooner, M.D.

London School of Hygiene and Tropical Medicine

It seems worth putting on record the procedure by which a strain of mumps virus was isolated in chick embryos in this country in 1948 from human saliva taken on the first day of the disease. Two other attempts in 1948 and 1949 failed. In one of these, saliva was not collected until the third day of illness; in the other it was collected on the first day but kept 24 hours in a domestic refrigerator. In both of these unsuccessful attempts, the amniotic route alone was used.

Isolation of strain BRU

Saliva was collected on 1st May, 1948, into 18 ml. of meat infusion broth in a 25 ml. screw-capped bottle. After light centrifugation, 3 ml. of supernatant was mixed with 1 ml. of broth containing 10,000 units of penicillin. A broth culture made from this mixture and incubated aerobically at 37° C. yielded no bacterial growth in one week.

Five nine-day eggs (Eggs Nos. 1–5) received 0·5 ml. each into the yolk sac, and five more were inoculated into the amniotic sac with volumes of 0·2 ml. (Eggs Nos. 7–10) or 0·1 ml. (Egg No. 6). The eggs were then incubated at 35° C. and opened on the fourth day. Four of the five "yolk sac" and three of the five "amniotic" embryos were alive. The amniotic fluid from one live egg (Egg No. 10) showed a trace of haemagglutination when tested by addition of about 0·05 ml. to 0·5 ml. of 0·5 per cent. washed fowl cells. Fluids from Eggs Nos. 6, 7, 8 and 9 showed no haemagglutination; the yolk sacs of Eggs Nos. 1–5 were not tested.

Fragments of yolk sac from each of the living eggs Nos. 1, 2, 3 and 4 were ground in a Griffith tube with amniotic fluid from two of the "amniotic" eggs (Eggs Nos. 9 and 10), including that which had shown trace haemagglutination. 0·5 ml. of the resulting suspension was inoculated into the yolk sacs of two more six-day eggs, which were incubated for a further five days at 35° C.; both were heavily contaminated with bacteria and discarded.

Further fragments of three of the yolk sacs (Eggs Nos. 1, 2 and 3) were ground in amniotic fluid of Egg No. 9, which had shown no haemagglutination; after centrifugation at 3,000 r.p.m. for 10 minutes, 0·1 ml. was injected amniotically into each of eight ten-day eggs, 500 units of each of penicillin and streptomycin being injected in a separate 0·1 ml. volume into the amniotic sacs of four of them. When these eggs were opened five days later, two, of which one had received antibiotics and one had not, yielded haemagglutinating amniotic fluids and failed to give bacterial growth on a blood agar plate.

Passage of 0·2 ml. of the pooled amniotic fluids of the two " positive " eggs into six nine-day eggs amniotically yielded one haemagglutinating amniotic fluid (Egg No. 22) which, however, was contaminated with cocci.

Fluid from Egg No. 22 inoculated, together with 500 units of penicillin, into three other eggs, yielded one, Egg No. 32, which was dead on harvesting, but the amniotic fluid of which haemagglutinated and passed the sterility test. This fluid was used for the fifth passage, at which, of six eggs inoculated, three survived to the fifth day, and all of these yielded haemagglutinating fluids. The allantoic fluid from one of these eggs also haemagglutinated.

Subsequent passages were very irregular in their yield of virus-bearing eggs, but the strain has now been maintained through 32 passages. After 15 amniotic passes, it was successfully grown in the allantoic cavity in which it has now been passed 17 times.

Properties of the BRU strain

The identification of the virus as a strain of mumps has been checked from time to time by serological tests with convalescent human sera and by comparison with the Enders American stain. No antigenic differences have so far been detected by either complement-fixation or haemagglutinin inhibition tests between crude infected allantoic fluids of the BRU strain and of the Enders American strain. The BRU strain has, however, remained much more erratic in its behaviour in eggs, so much so that infectivity titrations have in general proved useless. The only other point of difference between the two strains which has so far been discovered is in the resistance of the haemagglutinin to heat. If, in the form of 1 ml. of undiluted allantoic or amniotic fluid, the viruses are heated for 10 minutes at 50° C., the haemagglutinating power of BRU is destroyed or nearly destroyed while that of the American strain is affected little if at all.

The virus has survived for three years freeze-dried in the form of amniotic fluid diluted with an equal volume of 14 per cent. sucrose solution.

Summary

A strain of mumps virus isolated by egg inoculation in 1948 resembles the Enders American strain in haemagglutinin and complement-fixation tests done with crude egg fluids, but differs from it in the lower heat resistance of its haemagglutinin.

The irregular behaviour of mumps virus in eggs makes it advisable to use generous numbers of eggs when isolating a strain from saliva.

I wish to acknowledge with gratitude the assistance of Dr. C. M. Chu in the early stages of the isolation and the generosity of Dr. J. F. Enders in sending me his American strain. Dr. F. Fulton and Dr. C. L. Greenbury did the complement-fixation tests with these viruses.

Part of this work was done with the aid of a grant from the Medical Research Council.

AN ATYPICAL CASE OF SALMONELLA DUBLIN INFECTION

G. V. Purnell, M.B., Ch.B., Department of Clinical Pathology,
Manchester Royal Infirmary

This case is of interest because of the isolation of *Salm. dublin* from a prepatellar bursa of a patient who had no history of gastro-enteritis and who had not apparently been associated with an outbreak of food poisoning.

Case history

The patient, a woman aged 71, was first seen in the out-patient department on the 28th September, 1951, complaining of a painful swollen right knee. Two weeks previously she had slipped and fallen heavily on this knee; apart from the pain at the time of injury, she paid no further attention to it till a week later when the knee became swollen and stiff. There was a temporary improvement, but the pain and swelling increased the day before she was seen in the out-patient department. A purulent prepatellar bursitis was diagnosed and thick pus was aspirated. The patient was admitted to a surgical ward where the bursa was incised to facilitate drainage. It healed rapidly and she was discharged two weeks later.

Bacteriology

Numerous pus cells and occasional Gram-negative bacilli were seen in direct smears of the pus, and cultures on blood agar and MacConkey's medium produced a moderate growth of a Gram-negative bacillus. This organism was sluggishly motile and gave the biochemical reactions of the salmonella group. Slide agglutination reactions were indefinite, but a suspension of the organism was agglutinated to a titre of 1/50 by *Salm. typhi* O, *Salm. paratyphi B* O, and *Salm. enteriditis* H sera, but not by the patient's serum. The organism was sent to the Salmonella Reference Laboratory at Colindale for identification and Dr. Joan Taylor reported its antigenic structure to be IX, XII: g, p.——., and that it was therefore *Salm. dublin.*

Agglutination tests with the patient's serum, taken early in the illness, gave negative results, but 12 days later a further test made with the standard suspensions gave the following results:—

	H	O
Salm. typhi	Negative at 1/20	Positive at 1/640
Salm. paratyphi A	,, ,, 1/20	
Salm. paratyphi B	,, ,, 1/20	Positive at 1/640
Salm. enteriditis	Positive ,, 1/2,560	
Composite Salmonella	Negative ,, 1/20	

The organism was now agglutinated to a titre of 1/2,560 by the second sample of the patient's serum.

The patient's faeces were cultured on five occasions with the use of MacConkey and deoxycholate agar, and selenite and tetrathionate enrichment media, but salmonella organisms were not grown. After she had returned home, several specimens of faeces from the patient and her relatives were sent to the Public Health Laboratory, Manchester, for examination, but no salmonella organisms were isolated.

Discussion

An interesting feature of this case is that the patient could not recall any illness or symptoms of food poisoning either before or after the injury to her knee. She lives on the outskirts of Manchester with her husband and daughter, neither of whom had had any symptoms of food poisoning. Further inquiries about the family and their friends, including the source of their milk supply, failed to indicate how the infection had occurred.

Few outbreaks of food poisoning due to *Salm. dublin* are described in the British literature, and in the reported outbreaks milk has usually been the proven vehicle. Smith and Scott (1930) suggested that the organism was of bovine origin, and Lütje (1939) found that 404 of 409 strains of *Salmonella* isolated from a series of cattle were *Salm. dublin*. More recently Field (1948, 1949) published the results of extensive surveys of salmonella infection in cattle; and Smith and Buxton (1951) found that of 750 healthy cattle examined, 0·4 per cent. contained salmonellae in their faeces, the organism in every instance being *Salm. dublin*. Ritchie and Clayton (1951) isolated *Salm. dublin* from 10 per cent. of samples of faeces from 1,254 healthy Irish cattle but from only 3·7 per cent. of samples from 855 non-Irish cattle.

In the case now described the findings suggest that the patient had a subclinical infection due to *Salm. dublin* with transient bacteraemia; the coincident trauma to the prepatellar bursa resulted in the localization of the organism with the production of a suppurative lesion. The association of local trauma with abscess formation in typhoid infections has long been recognized, and is thought to be due to the organisms finding an area of tissue of lowered resistance. This association has been noted with the food-poisoning salmonellae, particularly *Salm. dublin*, but much less frequently. In most investigated outbreaks of salmonella infection symptomless excreters are found. In the report on food poisoning in England and Wales for 1950, 120 carefully studied family outbreaks were mentioned, in which 167 persons had symptoms and 172 were found to be symptom-free excreters (Report, 1951).

In the period 1941–48, five cases of unusual manifestations of *Salm. dublin* infection were reported in England and Wales, against eight due to all other salmonellae (Report, 1950*a*). These five comprised two cases of septicaemia, one of meningitis, one in which the organism was isolated from a liver abscess and one in which it was isolated from the peritoneal fluid of a woman who developed ascites in the puerperium. During 1949 *Salm. dublin* accounted for less than 2 per cent. of the total salmonellae isolated from outbreaks and sporadic cases, and yet of the 11 cases presenting unusual manifestations, six were due to *Salm. dublin* (Report, 1950*b*).

It is interesting to note that Jones (1951) reported a case in which *Salm. typhi* was isolated from a sebaceous cyst, first noticed two years previously. There was no previous history of typhoid infection.

Summary

The isolation of *Salm. dublin* from a prepatellar bursitis in a patient aged 71 who gave no history of food poisoning is described.

I wish to thank Dr. R. W. Fairbrother, Director of the Department, for his help and advice, and Dr. Joan Taylor for confirming the identity of the organism. I acknowledge also the technical assistance of Mr. Southall, F.I.M.L.T.

References

Field, H. I. (1948) *Vet. J.*, **104**, 251, 294, 323; (1949) *Vet. Rec.*, **61**, 109, 275.
Jones, A. C. (1951) *This Bulletin*, **10**, 247.
Lütje. (1939) *Dtsch. tierärztl. Wschr.*, **47**, 227, 246, 257.
Report (1950 *a*) *This Bulletin*, **9**, 148; (1950 *b*) *Ibid.*, **9**, 254; (1951) *Ibid.*, **10**, 228.
Ritchie, J. M. and Clayton, N. M. (1951) *Ibid.*, **10**, 272.
Smith, J. and Scott, W. M. (1930) *J. Hyg., Camb.*, **30**, 32.
Smith, H. W. and Buxton, A. (1951) *Brit. med. J.*, i, 1478.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The March Bulletin was issued on 24th March.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(15184) Wt. 8221—7283 3300 4/52 D.L./H.E./4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

MAY 1952 Vol. II

CONTENTS

Section I

Section II

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1

VACCINATION AGAINST SMALLPOX: INTERPRETATION OF RESULTS*

Maurice Mitman, M.D., F.R.C.P., D.P.H., Consultant Physician and Medical Superintendent, River Hospitals, Joyce Green, Dartford, Kent

SUMMARY:

Local reactions to vaccination are of two types, those due to *multiplication* of the living virus and those due to *sensitivity* to the virus, dead or alive, and to its products. The susceptible—the unvaccinated and those whose immunity from previous vaccinations has lapsed—respond to the living virus with the typical primary-type reaction which is maximal after the 7th day. The partially immune—those with some residual immunity from a previous vaccination—respond with a modified lesion which is called vaccinoid or accelerated. In fact, its onset is not accelerated and may even be delayed; but because it runs its modified course rapidly its maximum intensity is reached between the 3rd and 7th days. It should not be called an accelerated reaction. The sensitive subject, whether susceptible or immune responds to the virus, dead or alive, with an early reaction on the 2nd or 3rd day which has been called the immediate reaction of immunity, but is neither immediate nor a reaction of immunity. It has been regarded as the normal reaction of the immune subject, but as it may occur in those whose immunity has completely lapsed, it should not be called a reaction of immunity. Because sensitivity persists after immunity wanes revaccination commonly results in a combined early-and-vaccinoid reaction. Whilst the former is regressing the latter is developing, hence the impression that vaccinoid reactions start early. Distinction between sharp, early reactions and mild vaccinoid ones is sometimes difficult. Vesiculation, commonly regarded as a distinctive feature of vaccinoid reactions, occurs in sharp, early reactions although the vesicle is clinically different. No reaction at all has been regarded as evidence of complete immunity and of a refractory state, but it is most commonly due to errors in technique. Technical failures, even by the experienced, occur easily. On the other hand, some subjects are surprisingly easy to vaccinate: in a test series a single pressure, instead of multiple pressures, was successful in a high percentage of cases. Immunity following vaccination is shorter than is commonly believed. The best practice is to obtain a mimimal primary vaccination and to revaccinate at short intervals—one or two years in those subject to special risk. Even if revaccination does not take it probably boosts immunity, although serological evidence is required to support this hypothesis.

The object of this paper is to review the interpretations placed upon the local reactions to vaccination and revaccination against smallpox and to show that they need revising. For many years reactions were recorded as "successful" or "unsuccessful", or as "take" or "no take". This seemed simple and straightforward. Certain early reactions which appeared on the 2nd or 3rd day after revaccination and did not proceed to vesiculation were ignored. Later, enormous importance was attached to these very reactions. They were found to occur in highly immune subjects and were called Immediate Reactions or Reactions of Immunity. Subjects with some residual immunity, but not enough to prevent a "take", responded to revaccination with a local lesion which appeared and disappeared sooner than that following ordinary vaccination. This was the Accelerated or Vaccinoid Reaction; emphasis was placed as much on the rate and development of the reaction as upon its extent. Thus developed the classification which was widely accepted in international circles and in the armed Services, and which is summarised below:—

Time of Maximum Response	*Type of Response*	*Inference*
(1) After 7th day	Primary — typical vaccinia pustule.	No immunity or lapsed immunity.
(2) 3rd–7th day	Accelerated or vaccinoid ...	Some immunity
(3) 2nd–3rd day	Immediate reaction or reaction of immunity.	Immune
(4) —	No reaction	Failure of technique

* Paper presented to the Section of Epidemiology, Royal Society of Medicine, Feb. 15th 1952.

"No reaction at all" (4) was once regarded as evidence of "complete immunity" or of "insusceptibility to vaccination" or of "refractoriness". The danger of accepting such interpretations and of not allowing for failures of technique, which are quite common (*vide infra*) resulted in a swing the other way. It was then said that if an immediate, accelerated or primary reaction was not obtained the negative finding could not be accepted, even if repeated several times. In consequence, there developed a tendency to accept any redness, however slight, as a successful vaccination, occasionally with serious consequences. This paper is presented to emphasise that the immediate reaction of immunity is neither immediate nor a reaction of immunity; that the accelerated reaction is not accelerated, indeed its onset may be delayed: and that occasionally no reaction occurs in individuals immune to revaccination.

Types of Reaction. Reactions to vaccination fall into two primary classes:—

(*a*) Those which result from a inflammatory reaction due to multiplication of the living virus.

(*b*) Those due to sensitivity to the virus or its products.

If immunity is absent or low the living virus causes the reaction of *primary vaccinia,* with its maximum intensity after the 7th day and its progress through papule, vescile, and pustule to scab, and with local swelling, adenitis and constitutional disturbance from the viraemia. If there is some residual immunity the response is similar but *modified* and has been called *vaccinoid.* Its onset is not accelerated, indeed it may be delayed a little. There is no infectious disease in which the incubation period of attenuated attacks is shortened and it does not occur in vaccination. These modified lesions bear the same relation to primary vaccinia that the focal lesions of modified smallpox bear to the unmodified disease. In vaccinoid reactions the cutaneous lesion is milder, more superficial, and of shorter duration, and in this sense only is it accelerated. It may abort before maturing fully, and it leaves little or no scar.

The second type of reaction is not due to the living virus, but depends on sensitivity to the virus or its products. These reactions occur early—second or third day, or even earlier, but they are not immediate; if sharp, they last longer than is generally believed, e.g., seven days or more; they are not reactions of immunity although those subjects who show them have been vaccinated before and may have a considerable degree of immunity. *They may, however, occur in those whose immunity has completely lapsed and they can be produced with inactive or dead virus.* For this reason, the immediate reaction must not be regarded—as it has been—as the normal reaction of the *immune* subject to vaccination. It is the normal reaction of the *sensitive* subject, who may or may not be immune. Steps are being taken to eliminate the term "reaction of immunity" from official nomenclature.

Combined early ("immediate") and vaccinoid reactions. The proportion of revaccinated subjects who are sensitive to the virus or its products is high and the state of sensitivity persists long after immunity has lapsed; in many cases it never goes. It follows that a proportion of people whose immunity has waned will react to revaccination with an early reaction *and* a vaccinoid reaction, or if immunity has lapsed entirely, with an early reaction and a primary type reaction. Whilst one is regressing the other is developing.

In Figure 1 an attempt has been made to show diagrammatically that it is possible to have a combination of a sharp early and a mild vaccinoid, or a mild early and a sharp vaccinoid so little modified as to be a primary type, or

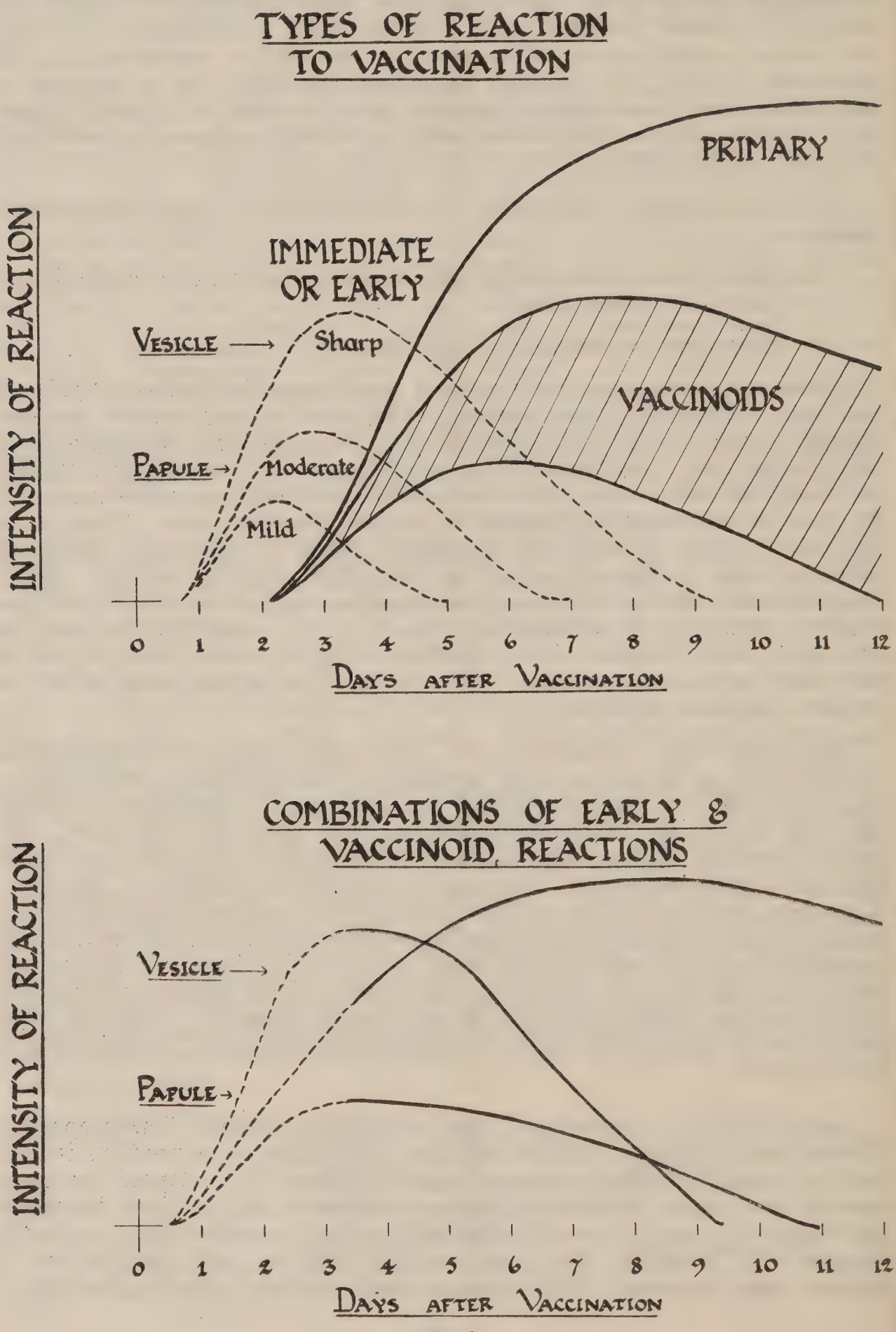


Fig. 1

a moderate early and a moderate vaccinoid. There is no parallel between the severity of the early and that of the vaccinoid reaction; indeed, the correlation if anything is negative. It can, therefore, be extremely difficult to distinguish between the sharp early reactions on the one hand and the vaccinoids or combinations of early and vaccinoids on the other. For this reason the percentages of the types of reactions given in the Table are subject to doubt.

RIVER HOSPITALS—JOYCE GREEN

Staff Vaccinations and Revaccinations, 1951

	1st Attempt			2nd Attempt			3rd Attempt			
	No.	Successful	Failures	No.	Successful	Failures	No.	Successful	Failures	
Primary Vaccinations	31	28	3	3	3	0	—	—	—	
Revaccinations	232	215*	17	9	7†	2	2	1‡	1	Plus 4 partial failures at 1st attempt, viz., one out of two insertions.
Totals ...	263	243	20	12	10	2	2	1	1	

Type of Reaction in:—	*215 successful Revaccinations	†7 successful 2nd Attempts	‡1 successful 3rd Attempt	4 Partial Failures i.e., only one of two insertions successful
Primary Type	c̄ 29%	3	—	1
Vaccinoid ...	c̄ 23%	1	—	1
Immediate ...	c̄ 48%	3	1	2

In distinguishing between sharp, early reactions and mild, vaccinoid ones it was once customary to regard vesiculation as the distinctive clinical feature of the vaccinoid reaction. But vesiculation can also occur in severe early reactions although it is clinically a different vesicle. It occurs earlier; it is very superficial; it is usually very small and does not spread laterally; it itches and is readily ruptured by scratching, producing an early and superficial scab which leaves no scar. This can occur in subjects with a high degree of vaccinal immunity, including the members of the staff of a smallpox hospital. The small vesicle which occurs in mild vaccinoid reactions, and which may be confused with the early vesicle, is simply a highly modified vaccinia vesicle. It is deeper than the early vesicle; it is papulo-vesicular in type, with a central umbilication; it appears later, spreads a little and may fuse with other vesicles; it lasts longer and, if associated with axillary adenitis and no itching, can be diagnosed with certainty as a vaccinoid reaction. In some instances a hand lens is needed to distinguish these points; but always the picture may be confused by the existence of a combined reaction.

Investigation. A short investigation into factors which determine the severity of early reactions has been conducted. During the outbreak of smallpox at Brighton a high proportion of severe early reactions was obtained

from the lymph then issued and the batch of lymph was regarded as a factor. An assurance was obtained that the potency of the lymph had not been deliberately stepped up. Comparisons were made of the reactions obtained with Government sheep lymph and a commercial calf lymph; and a further comparison was made with active lymph on the one hand and with lymph inactivated by heat and by teepol on the other hand. In every instance where comparisons were made the insertions of the lymph were made into the same arm of the subject. No material difference between calf lymph and sheep lymph was detected. It was confirmed that inactivated lymph was capable of causing early reactions, although they were not quite so marked as with the live virus. Despite these findings it was felt that at different times of the year there was an appreciable difference in the incidence of early reactions of a type severe enough to produce vesiculation, and the impression remained that this was in some way related to the batch of lymph.

The degree of sensitivity is naturally a factor in the severity of early reactions, but marked reduction in sensitivity can be induced by repeated revaccinations at short intervals: presumably some sort of desensitisation is induced.

Lastly, the quantity of material inoculated affects the severity and duration of early reactions. By comparing the reaction when the multiple pressures were spread over an area of 15 mm. diameter with one of 5 mm. diameter an appreciable difference was demonstrated, the intensity being greatest and lasting longer in the more concentrated area.

Absence of reaction. Perhaps the most interesting reaction to vaccination is no reaction at all. As stated above, "no reaction" has been attributed to complete immunity, to a refractory state and to poor technique or material. In all probability it can be all three. Although the intention of the investigation was to show that "no reaction" occurs in a proportion of persons who have full immunity without sensitivity it was not shown conclusively to be so. It is true that, as mentioned above, it is possible to reduce sensitivity by repeated revaccination at short intervals until virtually no reaction occurs, but in general it was found that in most cases "no reaction" was evidence of technical failure. The ease with which technical failure occurs is shown in the Table. Three out of 31 primary vaccinations failed and 17 out of 232 revaccinations. Of these 17, 9 were subjected to a second attempt and 2 again failed. It is of particular importance to note that 3 of the 7 successful second attempts responded with a primary-type reaction. It is worth recording that, on the worst day, 4 persons were vaccinated, 2 primaries and 2 revaccinations. Only 1 of the primaries took; at the second attempt all 3 failures showed reactions—the primary a typical primary reaction; one of the revaccinations, a mild vaccinoid reaction; and the other a sharp immediate reaction. These are high figures for an experienced vaccinator taking his time. It will be apparent that in the stress of an epidemic the proportion of failures of technique by inexperienced vaccinators must be higher.

Nevertheless, there are subjects who are partially or wholly refractory to vaccination; when they do react the lesion may be small and apparently modified, or very severe. The clinical impression obtained was that those whose skins were abnormal exhibited this feature more commonly than normals. Perhaps the explanation is to be found in the exceptional caution taken in vaccinating such subjects.

Whilst it is easy to *fail* to vaccinate, it can be very easy to succeed. All the test vaccinations of this series were carried out by the multiple pressure technique using 20 pressures per site. The multiple pressure method is simply a means of ensuring minimal penetration of the epidermis at multiple points so as to reduce the chances of failure. To prove this, attempts

were made to vaccinate and revaccinate by a single pressure of the needle —one, not 15 or 20. They were successful in a high proportion of cases. The vesicles produced were typical and small.

To reduce the reaction which could be attributed to secondary infection of the vaccination pustule, dressings penicillin tulle gras were applied except where there was a danger of sensitivity to the drug. But such local applications, with or without the occasional injection of penicillin, the general disturbance was reduced and the resultant scar was minimised. There is no evidence that this practice reduces the degree of immunity produced. Even if it were it is wiser to have minimal lesions at primary vaccinations and to revaccinate at shorter intervals. No evidence is available of the boosting value of revaccination in the immune but as serological investigations are now possible an attempt should be made to clear up this very important point.

PREVIOUS EXPERIENCE OF THE COMMON INFECTIOUS DISEASES, OF IMMUNISATION AND OF TONSILLECTOMY IN A GROUP OF SCHOOL CHILDREN

E. R. Bransby, Ph.D., M.Sc., Ministry of Health.

In September, 1947, an enquiry into the extent and causes of absence from school was begun under the aegis of the Ministries of Health and Education, in association with the Education and Health Authorities of Birmingham, Sheffield, Kesteven (Lincs.) and Worcestershire. The enquiry started at the beginning of the autumn term, 1947, and lasted one year.

The main findings of the enquiry—those relating to its primary object to ascertain the extent and causes of school absence—have already been published. (Bransby, 1951.) They are not discussed in this paper, which summarises information recorded by parents on cards sent to them, one for each child, before the enquiry began.

On the card the parent was asked to state whether or not the child had previously had diphtheria, measles, German measles, chicken pox, whooping cough, mumps and scarlet fever ; whether or not the child had been immunised against diphtheria, and whether or not the child had had a tonsillectomy operation.

All these recorded instances in the child's medical history have been analysed according to sex and age of the child and the social class of the *district* in which the school stands.

In Birmingham and Sheffield children attending schools in four kinds of districts were included: (*a*) a slum area scheduled for clearance ; (*b*) a working-class area not scheduled ; (*c*) a new housing estate ; (*d*) residential area.

The *school attended* was the basis of selection for inclusion, certain classes being chosen to participate. Six schools were in Sheffield, 41 in Birmingham, 12 in Kesteven and 14 in Worcestershire. The plan was to include about 1,000 children in each of the four districts of Birmingham and Sheffield and the same number in Kesteven and Worcestershire, that is, about 10,000 in all. Nine thousand four hundred and forty-four children were eventually included, 3,446 in Birmingham, 3,825 in Sheffield, 1,076 in Worcestershire and 1,097 in Kesteven.

Previous Experience of the Common Infectious Diseases

Table I shows by age groups the percentage of boys and girls in the different areas who, before the enquiry began, had had the various common infectious diseases. Also included in the Table is comparable information obtained from the Report of the School Epidemics Committee of the Medical Research Council (1938), from a study made in boarding and day public schools in the five years 1930-1934.

I. Percentage of Children who had had certain Infectious Diseases

	Boys																				
	Diphtheria			Measles			German Measles			Chicken Pox			Whooping Cough			Mumps			Scarlet Fever		
	Age Group			Age Group			Age Group			Age Group			Age Group			Age Group			Age Group		
	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C
Kesteven	0	0	2	44	77	78	13	21	29	24	40	49	50	58	49	7	34	48	2	6	6
Worcestershire	0	1	3	64	89	84	8	21	20	31	50	53	41	63	52	13	31	43	3	8	9
Birmingham Districts. Poor	4	7	16	81	86	81	11	16	18	38	50	40	48	61	47	18	35	37	6	10	13
Working Class	1	8	11	75	87	86	17	20	20	37	55	60	47	53	55	14	39	48	8	12	10
New Housing	1	6	2	85	87	83	16	24	29	41	49	58	55	53	51	26	38	46	10	14	12
Good	2	3	3	80	88	97	17	31	18	44	71	74	55	61	62	28	44	43	8	12	19
Sheffield District. Poor	4	11	5	89	92	85	8	16	15	38	60	56	43	42	44	23	40	36	4	11	9
Working Class	1	3	4	86	91	90	15	31	30	54	74	72	60	52	43	12	40	42	7	24	16
New Housing	3	7	10	80	91	84	25	23	24	54	59	61	47	47	34	27	34	39	7	17	14
Good	0	2	6	73	93	83	19	31	24	38	73	62	57	61	62	22	42	52	8	15	21
M.R.C. Report Public Schools (Boarding) } before entry, usually	—			72·5			28·6			66·6			70·2			32·5			8·3		
(Day) } 13½ years	—			82·2			24·1			49·9			60·9			34·8			9·6		

		Girls																					
Kesteven		0	0	0	48	73	85	10	23	21	23	44	48	46	56	62	12	33	49	2	6	12
Worcestershire		1	0	1	67	87	92	7	21	29	25	56	61	50	62	64	10	32	39	4	7	12
Birmingham Districts.	Poor	2	6	9	89	89	88	14	16	11	39	49	50	60	52	62	16	35	44	5	10	7
	Working Class	1	5	5	78	94	92	22	25	21	44	52	56	51	60	65	18	41	45	3	15	13
	New Housing	3	3	7	85	92	90	22	20	25	52	56	62	59	60	59	29	40	50	8	9	13
	Good	1	1	6	79	93	100	25	36	21	54	71	70	68	68	77	19	36	47	8	15	21
Sheffield District.	Poor	5	9	20	89	94	95	15	13	16	50	63	63	49	48	51	26	39	50	5	15	12
	Working Class	2	4	5	85	92	82	13	35	43	56	75	65	62	55	54	10	35	46	7	21	23
	New Housing	3	3	9	78	95	89	21	17	24	47	69	62	50	53	57	27	42	51	6	12	23
	Good	0	1	6	81	93	95	24	37	29	45	73	68	57	63	79	19	42	47	7	18	6
M.R.C. Report																							
Public Schools (Boarding)	before entry, usually $13\frac{1}{2}$ years	—			63·1			22·1			57·1			67·3			21·9			7·1		
(Day)		—			64·0			22·4			53·0			66·3			22·6			9·0		

Age Groups. A = Up to 7 years.
B = 8—11 years.
C = 12 or more years.

Diphtheria. In the " good " districts of Sheffield and Birmingham the percentages of children who have had diphtheria ranged from 0-2, 1-3 and 3-6 for the three age groups, under 7, 8-11 and 12 or more years, the corresponding figures for the poor districts being 2-5, 6-11 and 5-20. The rate was high in the new housing district of Sheffield. In Kesteven and Worcestershire the rate was of the same order as that in the good urban areas.

Measles. 78 per cent. of the children under 7 years had had measles ; 89 per cent. of those under 11 years, and 88 of those over 12. Among children up to 7 years the sickness rate tended to be somewhat greater in the poor than in the good districts ; this, however, was not so for older children. The rate in Kesteven was consistently lower than in other areas.

German Measles. The average rates were 16 per cent., 24 per cent., 23 per cent. at under 7 years, 8-11 years and 12 or more years respectively There was a striking social gradient in Sheffield and Birmingham. Thus the rates in the poor areas for children up to 7 years, 8-11 years and 12 or more years were 8-15 per cent., 13-16 per cent. and 11-18 per cent., the corresponding figures for the good areas being 17-25 per cent., 31-37 per cent. and 18-29 per cent. The rate in the new housing area in Sheffield was relatively low.

Chicken pox. At ages under 7 years, 8-11 years and 12 or more years the average rates were 42 per cent., 60 per cent. and 59 per cent. There was a consistent social gradient in Birmingham and a less marked gradient in Sheffield. Thus in Birmingham for children aged 12 or more years the rate was 40 per cent. among boys and 50 per cent. among girls in the poor district compared with 74 per cent. and 70 per cent. in the good district.

Whooping cough. The average percentage of children who before 7 years had had whooping cough was 53 ; of those between 8 and 11 years, 56, and of those over 12, 56. Both in Sheffield and Birmingham the rate was higher in the good districts than in the poor districts. Thus for children up to 7 years it was 43-60 per cent. in the poor districts compared with 55-68 per cent. in the good districts ; for children of 8-11 years, 42-61 per cent. and 61-68 per cent. ; and for children aged 12 or more years 44-62 per cent. and 62-79 per cent. The rate among girls tended to be somewhat greater than that among boys.

Mumps. The rate averaged 19 per cent. for children under 7 years, 37 per cent. at 8-11 years and 45 per cent. at 12 or more years.

Scarlet fever. The average rate was 6 per cent. at under 7 years, 13 per cent. at 8-11 years and 13 per cent. at 12 or more years. The rate was higher in the poor districts than in the good districts.

History of Previous Tonsillectomy

Parents, being asked to record whether or not the child had had a tonsillectomy operation, presumably took this to include adenectomy ; separate adenectomy generally forms only about 5 per cent. of these throat operations on school children.

Table II shows the percentages of children who had had tonsillectomy. For all the areas together 16·5 per cent of boys under 7 years of age had had the operation, the figure for girls being 13·9. For boys and girls aged 8-11 years the percentages were 26·3 and 22·0 respectively, and for those aged 12 or more, 22·3 and 30·4. The tonsillectomy rates in Kesteven were low, and those in Worcestershire were lower than those in Birmingham and Sheffield. The rates in the poor areas of Sheffield and Birmingham tended to be somewhat lower than those in the better class areas.

II. *Percentage of Children who had had a Tonsil Operation*

Age Group	Kesteven	Worcester-shire	Sheffield				Birmingham			
			Districts				Districts			
			Poor	Working class	New housing	Good	Poor	Working class	New housing	Good
Boys Up to 7 years ...	7	6	14	21	20	20	16	16	24	21
Girls „ „ ...	3	5	16	15	19	18	10	17	18	18
Boys 8–11 years ...	14	15	25	35	25	30	21	33	39	36
Girls „ „ ...	11	16	22	29	24	26	20	28	22	26
Boys 12 or more years ...	8	15	26	27	25	28	20	34	16	22
Girls „ „ „ ...	14	21	24	26	29	24	19	32	20	30

III. *Percentage of Children who had been Immunised against Diphtheria*

Boys Up to 7 years ...	67	83	76	92	92	91	92	88	95	97
Girls „ „ ...	70	87	81	93	88	92	92	94	93	97
Boys 8–11 years ...	76	89	84	88	88	90	83	87	90	99
Girls „ „ ...	79	89	86	95	94	93	83	88	95	96
Boys 12 or more years ...	80	85	87	90	88	86	62	84	86	87
Girls „ „ „ ...	91	94	76	98	93	84	83	77	84	95

Discussion

The data in Table I are obviously subject to errors of memory or misunderstanding which vary between districts and social classes ; some of the infectious diseases are now so slight and the symptoms so inconspicuous that parents may not realise that the child has had the disease ; the extent to which parents consult a doctor when a child has relatively mild symptoms varies from district to district. Measles is generally unmistakable, but in mild attacks of the other infectious diseases the children may have been seen by doctors in some cases and not in others. How important these factors are, is impossible to say, and Table I is presented with these limitations in mind.

The highest recorded sickness rates were for measles, followed by chicken pox and whooping cough, then by mumps, German measles, scarlet fever and finally by diphtheria. This is the usual order of frequency. By the time children reached the age of 12 or more, 88 per cent. had had measles, 59 per cent. chicken pox, 56 per cent. whooping cough, 45 per cent. mumps, 23 per cent. German measles, 13 per cent. scarlet fever and 6 per cent. diphtheria. These averages conceal interesting inter-class differences. Thus the rates for diphtheria and measles (among young children) were greater in the poor districts than in the good districts, but for German measles, chicken pox and whooping cough the reverse was the case. The three last-mentioned infections, often being (or regarded as being) trivial, are more likely to be remembered and recorded in the good districts. The only noticeable sex difference was the higher rate of whooping cough among girls than among boys. The high diphtheria rates in the poor districts may, to some extent be due to the slightly lower immunisation rates in the poor than the good districts. These are shown in Table III. The higher measles rate found among young children in the poorer districts agrees with previous observations (for example Halliday, 1928) and may be due to mixing earlier with other children, and thus being earlier exposed to infection.

Comparison between the results obtained by the School Epidemic Committee (Medical Research Council, 1938) for day and boarding public schools and those obtained in the present enquiry can only be made for children aged 12 or more years. The results for the children attending day public schools are most suitable for comparison with those for the children attending schools in the good neighbourhoods of Birmingham and Sheffield. The measles rates for boys were not greatly different between the two groups, but for girls the rates in the present enquiry were substantially above those for the day public schools. The rate for German measles was much the same. For whooping cough, the rates for boys were about the same, but for girls more, in the present enquiry. In both enquiries the rates for girls exceeded those for boys. For chicken pox, mumps and scarlet fever the rates found in the present enquiry were substantially greater than those found for the day public schools. In considering the results from the two enquiries, it must be remembered that the two groups of children belonged to widely different social classes which differ in the frequency with which they call in the doctor. There is no doubt that the infectious diseases were diagnosed by doctors more often in the public school group than in the council school group. Moreover, the two enquiries were separated by an interval of about 20 years, and during that interval the incidence and severity of certain of the diseases may have changed.

That there are extraordinary variations in the proportions of school children subjected to tonsillectomy in different (and often neighbouring) areas is well known. The percentages shown in Table III are consistent with those which might be expected if the annual tonsillectomy rates* be multiplied

* This Bulletin **9**, 1950, March, 26. p. 62.

by the child's years of school life. It is also well known that there is an extraordinary social gradient in tonsillectomy, the percentage of well-to-do children who have been tonsillectomised before entry to the public schools being as a rule some three times higher than that of children of the same age at local authority schools.

Summary

Analysis has been made of the information provided in the autumn of 1947 by parents of school children on whether or not the child had previously had diphtheria, German measles, chicken pox, whooping cough, mumps and scarlet fever; whether or not the child had been immunised against diphtheria, and whether or not the child had had a tonsillectomy operation. The data relate to 9,444 children attending schools in Kesteven (Lincs.) and Worcestershire, and four different kinds of districts, namely, a (*a*) slum area scheduled for clearance, (*b*) working-class area not scheduled, (*c*) new housing estate, (*d*) a residential area, in Birmingham and Sheffield.

The highest recorded rates were for measles followed by chicken pox and whooping cough, then by mumps, German measles, scarlet fever, and finally by diphtheria. By the time children reached the age of 12 or more 88 per cent. had had measles, 59 per cent. chicken pox, 56 per cent. whooping cough, 45 per cent. mumps, 23 per cent. German measles, 13 per cent. scarlet fever, and 6 per cent. diphtheria.

The rate for diphtheria and measles (among young children) was greater in poor districts than in the good districts, but for German measles, chicken pox, whooping cough, the reverse was the case. The only noticeable sex difference was the higher rate of whooping cough among girls than among boys.

Comparison of the rates for children aged 12 or more years attending schools in residential districts of Birmingham and Sheffield with the rates for children of the same age attending day public schools about 20 years ago showed that the rates for measles and whooping cough among boys, German measles among boys and girls found in the two enquiries were much the same. The measles and whooping cough rates for girls in the present enquiry were higher than those in public schools. For chicken pox, mumps and scarlet fever the rates in the present enquiry were substantially greater than those found for the day public schools.

For all the areas together 16·5 per cent. of boys under 7 years of age had had an operation for tonsillectomy, the figure for girls being 13·9 per cent. For boys and girls aged 8-11 years the percentages were 26·3 per cent. and 22·0 per cent. respectively, and for those aged 12 or more 22·3 per cent and 30·4 per cent.

References

Bransby, E. R. (1950) Med. Off. 1st and 8th Dec. 1951.
Halliday, J. L. (1928) Med. Res. Counc. Spec. Rep. Ser. No. 120, H.M.S.O.
School Epidemics Cte. Med. Res. Counc. (1938) Spec. Rep. Ser. No. 227, H.M.S.O.

Acknowledgments

The data on which this paper is based were collected during the course of an enquiry into school absence made in association with the Education and Health Departments of Kesteven, Worcestershire, Birmingham and Sheffield. I am very glad to express my appreciation of the co-operation of Dr. H. M. Cohen, Dr. M. C. Taylor, Dr. Wyndham Parker, Dr. T. W. P. Golby and Mr. R. E. Cousens, and to thank the teachers, health visitors and education welfare officers who collected the data.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, APRIL, 1952

(Issued from the General Register Office, Somerset House, W.C.2.)

	April 5	April 12	April 19	April 26	Average weekly figures for April 1951
Scarlet Fever	1,361	1,137	1,043	796	925
Whooping Cough	3,004	2,308	2,800	2,946	3,749
Diphtheria	22	24	32	35	39
Measles, excluding Rubella ...	6,836	5,476	6,775	6,137	22,137
Acute Pneumonia	711	682	830	747	657
Meningococcal Infection ...	60	44	48	46	39
Acute Poliomyelitis (Paralytic) ...	15	14	15	22	15
,, ,, (Non-Paralytic)	4	3	8	7	6
Ophthalmia Neonatorum ...	38	32	44	49	41
Puerperal Pyrexia and Puerperal Sepsis	221	244	279	281	73
Dysentery	548	489	327	275	895
Paratyphoid	7	13	9	8	14
Typhoid	1	1	2	—	6
Smallpox	7	4	2	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1

BIOCHEMICAL METHODS FOR BACTERIOLOGY

by Patricia H. Clarke, B.A., and S. T. Cowan, M.D.

National Collection of Type Cultures, Colindale Avenue, London, N.W.9

[This paper was published in the Journal of General Microbiology, 1952, **6**, 187-197, but in view of the wide interest taken in it it is reprinted here by kind permission of the Editors and Proprietors of the Journal.]

SUMMARY:

A series of biochemical methods using heavy suspensions of organisms and chemically-defined solutions are described; they include fermentations, reduction of nitrate and of methylene blue, production of indole, hydrogen sulphide and acetoin, and hydrolysis of gelatin, starch and urea. The tests use the preformed enzymes of the bacterial cells and the results are not complicated by side effects or the multiple reactions that occur in cultures growing in a nutrient medium containing the test substrate.

Methods developed by biochemists are seldom used in the characterization of bacterial species. Bacteriologists' tests are normally made in undefined media and the end-result often is a balance of opposing reactions; this applies particularly to the fermentation tests in which acid production from the breakdown of "sugar" must exceed alkali produced from the breakdown of protein constituents. Davis (1939) added washed bacterial suspensions to powdered sugars and showed that these non-multiplying suspensions gave clearer acid production than the same organisms growing in a nutrient medium containing the sugar. We applied the principle of testing for preformed enzymes in washed suspensions to other biochemical tests and, to avoid the need for massive growths, developed methods using small volumes of reagents.

Micromethods have recently gained favour with bacteriologists (Arnold & Weaver, 1948; Cook, 1948; Elek, 1948; Hannan & Weaver, 1948; Brough, 1950; Galton, Hardy & Mitchell, 1950; Morse & Weaver, 1950; Bachmann & Weaver, 1951; Fabrizio & Weaver, 1951; Hargrove & Weaver, 1951), but nearly all depend on a heavy inoculum and the growth of the organisms in a small volume of medium containing the test substrate. In the development of our methods speed has not been the determining factor, but a reasonably quick result has advantages in that a sterile technique is unnecessary. Chemically clean glassware is essential.

METHODS

Suspensions

Bacteria were grown on a favourable solid medium, preferably without blood or fermentable substance. Incubation was at the optimal temperature and for the shortest time (usually 18-24 hr.) to produce a good growth. A suspension was made by washing the growth from the slope or plate in tap water; this was spun to deposit the cells, and the deposit resuspended in a small volume of water. Suspensions made from blood agar cultures were washed once to remove haemoglobin. For a series of 20 "capillary sugars" and the full range of biochemical tests to be described, 1-1·5 ml. of heavy suspension was required. The number of slopes or plates of each culture incubated depended on the amount of harvested cells to be expected

and the number of tests to be made. Nutrient agar slopes, 100 mm. long in 150 × 20 mm. tubes, were used for most organisms ; 1-2 slopes of vigorously growing bacteria such as coliforms, pseudomonads and staphylococci normally provided ample growth, but 3-10 slopes or 1-4 plates of serum or blood agar were needed for organisms such as *Streptococcus pyogenes, Clostridium tetani* and *Corynebacterium hofmannii.*

Effect of suspension concentration and age of cultures

Suspensions were made as dense as the growth would allow, but there was a minimum opacity equivalent to a total count of *c.* 1×10^{11} cells/ml. below which the reactions became slower, weaker or uncertain. Dilution of the suspension and the age of the culture used in its preparation affect the speed and intensity of reactions. This is illustrated by the results obtained with *Pseudomonas hydrophila* (Table 1). Viable counts (Miles & Misra, 1938) on *Ps. hydrophila* suspensions showed that 10^8 organisms/ml. were inactive ; from

TABLE 1. *Effect of* (*a*) *age of culture and* (*b*) *suspension concentration on glucose fermentation and biochemical tests shown by* Ps. hydrophila (NCTC 7810)

Test	Time of reading (hr.)	Age of culture: 24 hr. Concentration of suspension 10^{12}	24 hr. 10^{11}	24 hr. 10^{10}	48 hr. 10^{12}	48 hr. 10^{11}	48 hr. 10^{10}	72 hr. 10^{12}	72 hr. 10^{11}	72 hr. 10^{10}
		Acid formation								
Glucose fermentation	1	A	A	0	A	0	0	0	0	0
	2	A	A	0	A	A	0	0	0	0
	4	A	A	A	A	A	0	A	0	0
	24	A	A	A	A	A	A	A	A	A
		Score in test*								
Indole formation ...	½	6	3	1	6	3	0	3	2	0
	1	8	8	3	6	6	1	6	3	0
	2	10	10	3	10	10	3	10	6	1
Nitrate reduction to nitrite	½	10	3	0	8	0	0	2	0	0
	1	10	3	2	8	1	0	3	0	0
Acetoin formation ...	½	3	0	0	1	0	0	0	0	0
	1	6	0	0	3	0	0	0	0	0
	2	8	2	0	8	0	0	0	0	0
	4	10	6	0	10	1	0	3	0	0
H_2S formation ...	1	6	0	0	3	0	0	2	0	0
	2	6	0	0	6	0	0	2	0	0
	4	8	0	0	8	0	0	3	0	0
Gelatin hydrolysis ...	4	8	6	0	8	6	0	6	3	0
	24	10	10	3	10	10	0	10	10	0
Starch hydrolysis ...	4	0	0	0	0	0	0	0	0	0
	24	8	6	0	6	3	0	6	6	0

A = acid formed.

* Numbers represent relative strength of reaction: 10 = maximum; 3 = definite positive; 2 and 1 = trace reactions. 0 = no reaction.

this it is clear that unsterilized glassware and tap water are not likely to introduce false reactions.

Effect of medium. The medium on which the culture is grown is important only if it encourages the development of an adaptive enzyme or if it inhibits the production of certain enzymes. A strain of *Bacterium dispar* took 8 days to ferment 1 per cent. lactose in peptone water ; suspensions made from growth on nutrient agar took 48 hours to ferment 5 per cent. lactose in our

microtest, but suspensions made from lactose agar fermented lactose in 24 hours. The effect of growth on glucose agar will be considered in connexion with the tests for acetoin and indole.

Buffer is desirable to prolong enzyme action but excess will slow the reactions ; 0·025M-phosphate buffer was found to be the optimal concentration for those tests in which this effect was studied.

Volumes were measured with dropping pipettes (Donald, 1913) but are only approximate as allowance was not made for the variation in drop-size of different solutions.

Fermentations

Three methods were tried and each showed that most bacteria have greater fermentative ability than is shown by the usual cultural tests in nutrient sugar media. Unfortunately, the three ways of testing our suspensions for preformed fermentative enzymes do not give identical results. Each method gives consistent results but only an extended trial and further experimental work will show which gives the best indication of the enzymic capacity of the test organism.

" Sugar " solutions. Glucose, xylose, lactose, sucrose, maltose and glycerol were made up as 25 per cent. (w/v) solutions. Sugars with lower solubilities were made up as follows: galactose, 20 per cent.; raffinose, 10 per cent.; starch, dextrin, mannitol, dulcitol and salicin, 5 per cent.

Buffer-indicator solution (BI). To 20 ml. 0·025M-phosphate buffer (pH 6·8) was added 1·0 ml. 1 per cent. ethanolic bromcresol purple. Both sugar solutions and buffer-indicator solutions were sterilized by momentary autoclaving (Davis and Rogers, 1939) or filtration.

Sugar-buffer-indicator solutions (SBI). Equal volumes of sugar solution and buffer-indicator solution were mixed in 65 × 10 mm. tubes. Mixtures were made up each week and when not in use were stored at 4°.

Method 1. Suspension (0·2 ml.), sugar solution (0·1 ml.), and BI (0·2 ml.) are mixed in 65 × 10 mm. tubes, and acid production observed after varying times in a 37° water-bath.

Method 2. Suspension and SBI are mixed in approximately equal volumes in a capillary tube 10 cm. long ; one end of the capillary is sealed and the other stuck in plasticine. Readings are made at intervals after incubation at 37°.

Method 3. Durham's tubes (35 × 8 mm.) are half-filled with 1 per cent. New Zealand agar + 0·1 per cent. bromcresol purple. These columns are stored at 4° until used. Two drops (0·04 ml.) sugar solution are pipetted on to the surface of each column, and 0·02 ml. suspension added. Columns are incubated at 37° and read up to 24 hours.

Capillary Tests

The next three tests were made in capillary tubes 10 cm. long. The tests are simple to carry out and to read, and comment seems unnecessary.

Catalase

" Ten vol." H_2O_2 is run into a capillary tube, followed by suspension. Gas is usually evolved immediately and only tubes not showing gas within 10 seconds are sealed for longer observation.

Methylene-blue reduction

Standardized methylene blue (British Drug Houses Ltd.) in concentrations of 0·1 and 0·01 per cent. are mixed with suspension and sealed. Readings are made after 4 and 24 hours at 37°.

Urease

Equal volumes of urea-buffer solution (urea 1 per cent., 0·0125M buffer pH 6·0, phenol red 0·00025 per cent.) and suspension are mixed. Sealed capillary tubes are incubated at 37° and read for alkali production after 4 and 24 hours.

Tests on Agar Columns

Gelatin hydrolysis

To detect gelatin hydrolysis Frazier (1926) flooded nutrient gelatin with acid mercuric chloride; this formed a thick white opalescence with the gelatin and left clear zones in areas of hydrolysis. Oakley, Warrack and Warren (1948) adapted the method to estimate gelatinases in bacterial toxins, using columns of 0·4 per cent. gelatin and 1 per cent. agar in water. For our microtest the columns were made up with 0·4 per cent. gelatin and 0·5 per cent. New Zealand agar. We found 0·5 per cent agar gave rather better results than 1 per cent.; when the gelatin concentration was less than 0·4 per cent. the opacity of the control tube was too faint. No difference was observed in the results for organisms grown on nutrient agar, serum agar, or 1 per cent. glucose agar. The mercuric chloride diffuses slowly through the agar, and tests should be left on the bench for at least 30 minutes before reading. The tubes were read against a blank gelatin-agar tube, and a positive reaction was indicated by a clear zone under the meniscus. (For a fuller description of the appearance of the tube, see Oakley *et al.* 1948.)

Cl. botulinum, Cl. histolyticum, Cl. sporogenes, Cl. welchii, Ps. hydrophila, Staphylococcus aureus and *Vibrio* spp. gave positive results at either 4 or 24 hours; no positive reactions were obtained from organisms negative by the cultural method.

Method. Suspension is prepared from a 24 hour culture grown on any suitable medium. For each test 0·04 ml. suspension is pipetted into each of two Durham's tubes containing 0·4 ml. gelatin-agar base. The tubes are placed in the air incubator at 37° and one is tested at 4, and the other at 24 hours. Acid mercuric chloride ($HgCl_2$, 15 g.; distilled water, 100 ml.; conc. HCl, 20 ml.) is added, and the tubes read after 30 minutes at room temperature against a blank gelatin-agar tube treated in the same way.

Starch hydrolysis

The microtest for starch hydrolysis is based on the same principle as that for gelatin. The suspension is placed on a column of starch-agar and, at the end of the test period, Lugol's iodine is added to test for the remaining starch. The optimum composition of the column was found to be 0·2 per cent. agar and 0·05 per cent. potato starch.

Positive reactions were obtained in 2 hr. with the more active starch-hydrolysing organisms, but we decided to use 4 and 24 hr. as the test periods, so that the weaker reactors could be detected. Positive results were obtained with *Cl. sporogenes, Ps. hydrophila, Shigella dysenteriae, Sh. flexneri* and *Vibrio* spp.

Method. Suspension is prepared from a 24 hr. culture grown on any suitable medium. For each test 0·04 ml. suspension is pipetted into each of two Durham's tubes containing 0·4 ml. starch-agar base. The tubes are placed

in the air incubator at 37° and tested at 4 and 24 hr. Lugol's iodine (2 per cent. potassium iodide, 1 per cent. iodine, in distilled water) is added and the tubes read after 30 min. at room temperature against a blank starch-agar tube treated in the same way.

Hydrogen sulphide

The production of H_2S from peptone media depends on the reduction of sulphur from sulphur-containing amino-acids or other sulphur compounds in the medium. We devised a micromethod for the detection of H_2S-production from cystine. Morse & Weaver (1950), in their microtest using ' thiopeptone ', obtained satisfactory results with lead acetate paper. In the small volumes with which we were working the amounts of H_2S formed were insufficient to be detected by this method. We attempted to carry out the test with a strand of cotton impregnated with lead acetate inserted at the top of a capillary tube, but this was not sensitive enough. Finally we made the test at the top of a column of lead acetate agar, where the H_2S formed was detected by the development of a brown or black colour at the interface. The lead acetate agar base (LAAB) was prepared by adding 1 ml. of a 0·05 per cent. solution of basic lead acetate (to which just enough HCl had been added to prevent precipitation) to 9 ml. 1 per cent. New Zealand agar in water. LAAB was more sensitive than ferric ammonium citrate agar. The cystine was made up in 0·1 N-HCl and adjusted before use to pH 7·4–7·6 with dilute NaOH. Phosphate buffer was not used as it did not improve the sensitivity and, by reacting with the lead acetate, made the test more difficult to read. The addition of sodium thiosulphate had no effect on the speed or intensity of the reaction. The optimum pH was not critical ; *Ps. hydrophila* gave positive reactions at pH values 6·0, 6·4, 6·8, 7·2, 7·6, 7·8 and 8, but the reactions were stronger and more rapid at pH 7·2–7·8.

With a heavy suspension of an H_2S-positive organism a black ring develops in 15–30 min. With most organisms the strength of the reaction and the rate at which it is produced depend on the concentration of the suspension. A 10^{12} organisms/ml. suspension of a 24 hr. culture of *Ps. hydrophila* produced a strong reaction in 30 min., but 10^{10} organisms/ml. gave no reaction in 24 hr. Heavy suspensions of most of the organisms we tested gave positive results in 1–2 hr., but we left the tubes overnight so that weak reactions could be observed. This method is rather more sensitive than the cultural method for the detection of H_2S from peptone water. Positive results were obtained with *Bact. aerogenes, Bact. coli, Cl. histolyticum, Cl. sporogenes, Proteus morganii, Ps. hydrophila* and *Salmonella typhi.*

Method. Suspension is prepared from a 24 hr. culture grown on any suitable medium. To columns of 0·4 ml. LAAB in Durham's tubes are added 0·04 ml. cystine solution (0·1 per cent. at pH 7·4) and 0·04 ml. suspension. The tubes are placed in the air incubator at 37° and read at intervals up to 24 hr.

Other Tests

Nitrate reduction

The reduction of nitrate to nitrite was detected with dimethyl- -naphthylamine (Wallace & Neave, 1927) and sulphanilic acid. The reaction was rapid with all the species tested ; at 30 min. the results were consistent with the usual cultural method. We did not have any false negatives produced by reduction of nitrite, but these could be detected by zinc dust (ZoBell, 1932).

Method. Suspension, 0·04 ml., is mixed in 65 × 10 mm. tubes with 0·05 per cent. $NaNO_3$, 0·06 ml. ; phosphate buffer (0·025 M, pH 6·8), 0·04 ml. After 30 min. at 37° (water-bath) 0·06 ml. dimethyl- α-naphthylamine solution

(6 ml./l. 5N-acetic acid) and 0·06 ml. sulphanilic acid (8 g./l. 5N-acetic acid) is added and read 5 min. later. A blank test on the nitrate and buffer is included with each batch of tests.

Acetylmethylcarbinol (acetoin)

Acetoin is formed from the breakdown of glucose by *Bact. aerogenes* and other bacterial species. Harden (1906) showed that it was the active substance in the Voges-Proskauer test; on the addition of strong potassium hydroxide the acetoin is oxidized to diacetyl which then condenses with substances in the medium containing a guanidine group to give a red colour. Acetoin can also be detected and estimated by oxidation to diacetyl and precipitation as the red nickel dimethylglyoxime (Lemoigne, 1920; Kluyver, Donker & Visser't Hooft, 1925), and estimated by iodine titration (Langlykke & Peterson, 1937). As the V.P. reaction is very sensitive and could easily be adapted to the microtechnique, we preferred a method derived from it. Glucose, creatine, and 0·025M-phosphate buffer (pH 6·8) were used as the substrate; glucose at 10 per cent. gave better results than 5 or 25 per cent. Creatine gave better results than 5 per cent. peptone, arginine, or guanidine nitrate, and there was no significant difference between adding creatine with the glucose or adding it with the test reagents, 5 per cent. ethanolic -naphthol (Barritt, 1936) and 40 per cent. KOH.

Medium and pH *value.* Suspensions of *Ps. hydrophila* from cultures grown 24 hr. on nutrient agar at pH 7·6 gave strong reactions after 1–2 hr. at 37° (water-bath), although *Bact. coli* and *Bact. aerogenes* were negative. After 24 hr. traces of acetoin could be detected with both *Bact. coli* and *Bact. aerogenes* suspensions. Strong reactions with *Bact. aerogenes* suspensions from nutrient agar were only obtained after 24 hr. in the presence of peptone, which may have allowed growth to take place. Suspensions of *Bact. aerogenes* from cultures grown 24 hr. on 1 per cent. glucose agar or acid agar (nutrient agar at pH 4) gave strong reactions in 30 min., while *Bact. coli* suspensions from glucose or acid agar behaved as those from nutrient agar (Table 2). *Ps. hydrophila,* tested after 30 min., gave stronger reactions when grown on glucose agar (Table 2).

TABLE 2. *Acetoin production by suspensions from growth on different media*

Glucose 10%, 0·02 ml.; creatine 0·2%, 0·02 ml.; buffer 0·04 ml.; suspension, 0·04 ml.

Time of test (hr.)	Suspension of culture: *Bact. coli* (NCTC 86)			*Bact. aerogenes* (NCTC 8348)			*Ps. hydrophila* (NCTC 7810)		
	Medium on which culture had been grown								
	N.A.	G.A.	A.A.	N.A.	G.A.	A.A.	N.A.	G.A.	A.A.
	Score in test*								
½	0	0	0	0	6	4	4	8	0†
1	0	0	0	0	10	10	nt	nt	nt
4	1	0	1	0	10	10	nt	nt	nt
24	2	1	0	2	10	10	10	10	nt

N.A.=nutrient agar; G.A.=glucose agar; A.A.=acid agar.
* Numbers 0 to 10 represent relative scoring of strength of reaction; see Table 1.
†=no visible growth on acid agar; nt=not tested.

Silverman & Werkman (1941) showed that the *Bact. aerogenes* enzyme system responsible for acetoin production from pyruvic acid was only formed when the organism was grown on an acid medium, and that crude enzyme expressed from the bacteria had a pH optimum of about pH 5.

A pH curve (Sørensen's phosphate buffers 0·025M or 0·06M, pH 4·5–8·0) with suspensions of *Bact. aerogenes* and *Ps. hydrophila* grown on glucose agar, showed an optimum on the acid side of the range. After 2 hr. the breakdown of glucose had produced enough acid to overcome the effect of the 0·06M buffer, and there was little difference in the results obtained in tubes with initial pH values between 4·5 and 7·2 (Table 3). We did not attempt to determine the pH optimum more accurately. For the microtests the greatest sensitivity was obtained by starting the reaction at a pH slightly higher than the optimum, and using a weak buffer. With no buffer, or if the reaction was started at a lower pH, the system became too acid. We therefore started our tests at pH 6·8 with 0·025M buffer.

Time. The optimum incubation period depends on the sensitivity required. At 24 hr. the test is more sensitive than the usual cultural methods, and *Bact. coli* suspensions are weakly positive. Again, after the reagents are added and the tubes shaken to aerate the solutions, the rate at which the colour develops depends on the amount of acetoin present. With very small quantities the colour development may be delayed for 1–2 hr. For the detection of minimal quantities the suspension-glucose mixture should be left at 37° for 24 hr. and the test read up to 1 hr. after the reagents have been added. For routine use we adopted an incubation period of 1 hr. and read the tests 10 min. after the addition of the reagents. Under these conditions the test clearly distinguished between *Bact. coli* and *Bact. aerogenes* grown on glucose agar.

TABLE 3. *Effect of* pH *value and buffer concentration on acetoin production by :*

(P) Ps. hydrophila NCTC 7810 *and by* (B) Bact. aerogenes NCTC 8348

Glucose 10%, 0·02 ml.; creatine 0·2%, 0·02 ml.; buffer 0·04 ml.; suspension 0·04 ml.

Organism	Time of test (min.)	0·025M phosphate buffer initial pH value†								0·06M phosphate buffer initial pH value†							
		4·5	5·6	6·0	6·4	6·8	7·2	7·6	8·0	4·5	5·6	6·0	6·4	6·8	7·2	7·6	8·0
		Score in test*															
P	5	3	2	2	2	1	1	0	0	3	3	1	1	1	1	1	1
	15	6	6	4	6	5	2	3	4	4	4	4	3	3	2	2	2
	30	8	8	8	8	8	8	7	7	6	6	6	6	6	4	3	3
	60	10	10	10	10	10	10	10	10	8	8	10	10	10	10	10	10
	120	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
B	5	2	1	1	1	1	1	1	1	1	1	1	1	2	1	1	0
	15	4	4	4	3	2	2	1	1	2	2	1	0	1	0	0	0
	30	6	6	8	8	7	6	6	6	3	3	3	3	1	1	1	1
	60	8	8	9	9	9	9	9	9	6	6	6	6	3	2	1	1
	120	10	10	10	10	10	10	10	10	7	8	8	9	9	10	5	4

* Numbers 0–10 represent relative scoring of strength of reaction; see Table 1.

† Initial pH value; fermentation products cause a decrease in pH value during the experiment.

Method. Suspension is prepared from a 24 hr. culture grown on 1 per cent. glucose agar, and 0·04 ml. suspension is added to a mixture of 10 per cent. glucose, 0·02 ml.; 0·2 per cent. creatine, 0·02 ml.; 0·025M-phosphate buffer (pH 6·8), 0·04 ml.; in a 65 × 10 mm. tube. After incubation in a water-bath at 37° for 1 hr., 0·06 ml. 5 per cent. ethanolic α-naphthol is added and the tubes shaken; 0·04 ml. 40 per cent. KOH is then added, the tubes shaken again, returned to the water-bath for 10 min., shaken again and read.

Indole

The production of indole from peptone depends on the presence of tryptophan in the medium. When the suspension from a culture grown on nutrient agar is incubated with tryptophan, indole is formed fairly rapidly and can be detected by any of the indole test reagents. Kovac's reagent was preferred to Böhme's as the results were easier to read and only one solution was needed. Kovac's reagent was made up in pure *iso*amyl alcohol, which Arnold & Weaver (1948) found gave better results than the other alcohols tested. In a dark bottle the solution remained stable on the bench for several months; the colour remained a golden yellow and no false colorations appeared in the test.

Tryptophanase is only formed when the organism is grown in the presence of tryptophan and in the absence of glucose. Evans, Handley & Happold (1941) showed that non-viable suspensions of cultures grown with glucose or on tryptophan-free medium would develop tryptophanase when left in contact with tryptophan for a short time.

In the microtest for indole, indole-positive organisms were grown on nutrient or serum agar and suspensions gave positive results in $\frac{1}{2}$–1 hr. *Bact. coli* and *Bact. aerogenes* were grown on glucose agar for the acetoin test, and these suspensions were used to check the effect of growth on glucose on the other microtests. Only the indole reaction was affected and, as Evans *et al.* (1941) found, the inhibition of the tryptophanase activity by *Bact. coli* was overcome after the suspension had been in the presence of tryptophan for 1–3 hr. (Table 4). Similar results were obtained with *Ps. hydrophila* and *Pr. morganii*. It is, therefore, possible to carry out all the microtests on suspensions from glucose agar cultures if the duration of the indole test is prolonged.

TABLE 4. *Indole production by suspensions from growth on agar and glucose agar*

Tryptophan 0·1%, 0·06 ml.; buffer, 0·04 ml.; suspension, 0·04 ml.

Time of test (hr.)	Suspension of culture: *Bact coli* (NCTC 86) — Medium on which culture had been grown: N.A.	G.A.	*Bact. aerogenes* (NCTC 8348) — N.A.	G.A.
	Score in test*			
0·5	6	0	0	0
1	8	1	0	0
1·5	8	3	0	0
2	10	6	0	0
3	10	10	0	0
4	10	10	0	0

N.A. = nutrient agar; G.A. = glucose agar.
* Numbers 0–10 represent relative scoring of strength of reaction; *see* Table 1.

For very light suspensions and when older cultures are used, an incubation period of 4–6 hr. for the test may be necessary, but for our usual test we have taken an incubation period of 1 hr. when the organism is grown on nutrient or serum agar and 2–3 hr. when grown on glucose agar. Among the limited number of organisms tested no positive reactions were obtained for cultures negative by the cultural methods. *Bact. coli, Cl. bifermentans. Ps. hydrophila, Pr. morganii* gave positive results.

Method. Suspension is prepared from a culture grown on nutrient agar. serum agar, or 1 per cent. glucose agar, and 0·04 ml. is added to a mixture of 0·1 per cent. DL-tryptophan, 0·06 ml. and 0·025M-phosphate buffer

(pH 6·8), 0·04 ml. The tubes are incubated in the water-bath at 37° for 1 hr. (3 hr. when suspension is from a glucose medium) and 0·06 ml. Kovac's reagent (*p*-dimethylaminobenzaldehyde, 5 g.; *iso*amyl alcohol, 75 ml.; conc. HCl, 25 ml.) added. The tubes are shaken and read immediately.

Discussion

The advantages of the methods described in this paper are simplicity of technique, clear-cut results, use of chemically-defined substrates instead of complex media, small amount of cells required to carry out a series of tests, rapidity and reproducibility. We have approached the bacterial cell as a system of preformed enzymes, and, using viable but non-multiplying suspensions, we tried to devise tests for the enzyme reactions used in bacterial characterization. With this in mind it is necessary to consider whether we want to assess the actual or the potential enzymic capacity of the cell. On nutrient agar at pH 7·6 *Bact. aerogenes* does not form the acetoin-producing enzyme, but it can be seen as a potential enzyme system because it is formed when the organism is grown on nutrient agar at pH 4–5, or on glucose agar. Again, the tryptophanase of *Bact. coli*, developed on nutrient agar, is inhibited when the organism is grown on a glucose medium. In this system the tryptophanase becomes active when the cells are in contact with tryptophan for about an hour. Therefore, if we want to realise the potentialities of the enzyme pattern, we may need to vary the conditions of our tests and to modify the medium on which the organism is grown. On the other hand, the ability to produce an enzyme on a particular medium may be of value in differentiating taxonomic groups; for example, the production of the acetoin enzyme by *Ps. hydrophila* on nutrient agar distinguishes it from *Bact. aerogenes*.

When the sensitivity of a test is increased, some organisms previously described as negative may be shown to give weakly positive reactions; this may arise either from an increase in the sensitivity of the test reagents, or from a change in the conditions of test. In this study only the H_2S, acetoin and fermentation tests gave greater sensitivity. For the H_2S test we devised a sensitive indicator, but with non-growing organisms, very heavy suspensions were needed. The detection of H_2S in culture media is made unsatisfactory by variation in media; in this laboratory we found that 0·1 per cent. cystine added to nutrient broth made each batch more consistently useful for H_2S production as detected by lead acetate papers (ZoBell & Feltham, 1934), but our microtest, like that of Morse & Weaver (1950), is even more sensitive.

Fabrizio & Weaver (1951) showed that most coliforms are able to produce some acetoin; this can also be shown by using highly sensitive test reagents (Batty-Smith, 1941) on 10 to 14-day cultures in glucose phosphate peptone medium (unpublished observations). We may have to decide between detecting all the positive reactors up to the sensitivity limit of our tests or choosing conditions which will give consistency with the accepted cultural method.

So far these tests have been worked out and applied to only a limited number of strains. If experience shows that we cannot resolve the discrepancies between the tests on growing cultures and on washed suspensions we shall not be able to apply the microtests to ordinary diagnostic work. However, our methods show more clearly than growing cultures the fundamental enzymic make-up of bacteria, and may show unsuspected relationships between different species or genera. As a systematic study, a re-characterization of species on the basis of enzymic patterns may be just as important and profitable as antigenic analysis.

References

ARNOLD, W. M., JUN. & WEAVER, R. H. (1948). Quick microtechniques for the identification of cultures. I. Indole production. *J. Lab. clin. Med.* **33,** 1334.

BACHMANN, B. & WEAVER, R. H. (1951). Rapid microtechnics for identification of cultures. V. Reduction of nitrates to nitrites. *Amer. J. clin. Path.* **21,** 195.

BARRITT, M. M. (1936). The intensification of the Voges-Proskauer reaction by the addition of α-naphthol. *J. Path. Bact.* **42,** 441.

BATTY-SMITH, C. G. (1941). The detection of acetyl-methyl-carbinol in bacterial cultures. A comparative study of the methods of O'Meara and of Barritt. *J. Hyg., Camb.* **41,** 521.

BROUGH, F. K. (1950). A rapid microtechnique for the determination of nitrate reduction by micro-organisms. *J. Bact.* **60,** 365.

COOK, G. T. (1948). Urease and other biochemical reactions of the Proteus group. *J. Path. Bact.* **60,** 171.

DAVIS, J. G. (1939). A rapid, simple and reproducible method for the determination of bacterial sugar fermentations. *Zbl. Bakt.* (2 Abt.), **101,** 97.

DAVIS, J. G. & ROGERS, H. J. (1939). The effect of sterilisation upon sugars. *Zbl. Bakt.* (2 Abt.) **101,** 102.

DONALD, R. (1913). An apparatus for liquid measurement by drops and applications in counting bacteria and other cells and in serology, etc. *Proc. Roy. Soc.* B, **86,** 198.

ELEK, S. D. (1948). Rapid identification of Proteus. *J. Path. Bact.* **60,** 183.

EVANS, W. C., HANDLEY, W. R. C. & HAPPOLD, F. C. (1941). The tryptophanase-indole reaction. 4. *Biochem. J.* **35,** 207.

FABRIZIO, A. & WEAVER, R. H. (1951). Rapid microtechnics for identification of cultures. IV. Acetylmethylcarbinol production. *Amer. J. clin. Path.* **21,** 192.

FRAZIER, W. C. (1926). A method for the detection of changes in gelatin due to bacteria. *J. infect. Dis.* **39,** 302.

GALTON, M. M., HARDY, A. V. & MITCHELL, R. B. (1950). The public health laboratory diagnosis of enteric infections. *Amer. J. trop. Med.* **30,** 77.

HANNAN, J. & WEAVER, R. H. (1948). Quick microtechniques for the identification of cultures. II. Fermentations. *J. Lab. clin. Med.* **33,** 1338.

HARDEN, A. (1906). On Voges and Proskauer's reaction for certain bacteria. *Proc. Roy. Soc.* B, **77,** 424.

HARGROVE, R. E. & WEAVER, R. H. (1951). Rapid microtechnics for identification of cultures. VI. Citrate utilization. *Amer. J. clin. Path.* **21,** 286.

KLUYVER, A. J., DONKER, H. J. L. & VISSER'T HOOFT, F. (1925). Über die Bildung von Acetylmethylcarbinol und 2, 3-Butylenglycol im Stoffwechsel der Hefe. *Biochem. Z.* **161,** 361.

LANGLYKKE, A. F. & PETERSON, W. H. (1937). The determination of acetylmethylcarbinol. *Industr. Engng. Chem.* (Anal. ed.) **9,** 163.

LEMOIGNE, M. (1920). Réaction spécifique du 2·3-butylèneglycol et de l'acétylméthylcarbinol, produits de la fermentation butylène glycolique. *C.R. Acad. Sci., Paris,* **170,** 131.

MILES, A. A. & MISRA, S. S. (1938). The estimation of the bactericidal power of the blood. *J. Hyg., Camb.* **38,** 732.

MORSE, M. L. & WEAVER, R. H. (1950). Rapid microtechnics for identification of cultures. III. Hydrogen sulfide production. *Amer. J. clin. Path.* **20,** 481.

OAKLEY, C. L., WARRACK, G. H. & WARREN, M. E. (1948). The kappa and lambda antigens of *Clostridium welchii*. *J. Path. Bact.* **60,** 495.

SILVERMAN, M. & WERKMAN, C. H. (1941). The formation of acetylmethylcarbinol from pyruvic acid by a bacterial enzyme preparation. *J. biol. Chem.* **138,** 35.

WALLACE, G. I. & NEAVE, S. L. (1927). The nitrite test as applied to bacterial cultures. *J. Bact.* **14,** 377.

ZOBELL, C. E. (1932). Factors influencing the reduction of nitrates and nitrites by bacteria in semisolid media. *J. Bact.* **24,** 273.

ZOBELL, C. E. & FELTHAM, C. B. (1934). A comparison of lead, bismuth, and iron as detectors of hydrogen sulphide produced by bacteria. *J. Bact.* **28,** 169.

(*Received by the Editors of the Journal of General Microbiology, 4th September,* 1951)

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.
Tel: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The April Bulletin was issued on 24th April.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(15568) Wt. 8221—7283 3300 5/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

JUNE **1952** **Vol. 11**

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the

Ministry of Health, Savile Row, W.1.

FOOD POISONING DUE TO THE CONSUMPTION OF SHELLFISH

J. Stevenson Logan, M.B., D.P.H., Medical Officer of Health, Southend-on-Sea C.B., and John O. Oliver, O.B.E., M.B., former Director of Pathology, Southend-on-Sea Hospitals and Honorary Director, Southend-on-Sea Laboratory (Public Health Laboratory Service).

At the beginning of September, 1949, there occurred food poisoning associated with the eating of shrimps, prawns and cockles, while illness following the consumption of cockles continued to be reported during the month. A total of 437 cases was reported ; 18 implicated shrimps, 21 prawns, and 398 cockles. The County Borough of Southend-on-Sea yielded 135 cases and many different places the rest.

The outbreak presented features which, at first, were not only difficult to explain, but tended to be misleading. The week-end of September 2nd-5th produced 100 cases (including all those associated with shrimps and prawns), for 94 of which the source of the food was identified. Altogether five suppliers were involved, but as all the cases, except 10 due to cockles, were associated with shed " H," attention was focused there.

Being guided by experience of a typhi-murium infection in 1946, the staff of this shed were investigated bacteriologically, *without finding anything significant*. The proprietor and an assistant, admitted having suffered from diarrhoea on the night of September 3rd-4th, and the employee had a septic spot on his hand. Only the cockles were processed in this shed, the shrimps being cooked elsewhere locally, and the prawns came via Billingsgate Market.

The next week-end, September 10th and 11th gave rise to 106 cases, but in only 52 of them could the source of supply be traced. This time only cockles were involved, and four sheds were implicated, viz., " B."—22 cases ; " G."—4 cases ; " H,"—2 cases ; " I."—24 cases ; sheds " B." and " I." became new centres for investigation.

It was now necessary to consider whether there was anything wrong with the industry as a whole, and urgent consultations took place between the officers of the local authorities and trade interests. As a result, it was agreed to change the gathering grounds, to discontinue the practice of dipping the cockles in the Leigh Creek immediately before unloading, to cook for seven instead of six minutes—a major economic concession which reduced the bulk of the fish by about 20 per cent.—to pay special attention to the cleansing of tanks and fish riddles, and generally to improve methods of handling in the sheds.

Meanwhile concurrent investigations had shown that there was no possibility of chemical contamination of the fishing grounds, and the cooking process, when properly and conscientiously carried out, produced a fish which was practically free from organisms when taken from the cooking pots.

One week later there was a third flare-up, when 46 cases came to light, the source of the fish being identified in only ten of them, i.e., shed " B." 3 cases ; shed " G." 7 cases.

By this time it was known that no organism ordinarily regarded as pathogenic had been isolated from any of the sufferers, and the resultant illness had nowhere proved grave. It seemed likely therefore that the illness was caused by non-specific organisms, which had proliferated to an unusual degree in the abnormally high and sustained temperatures which then prevailed.

Here mention may be made of several cases of food poisoning which occurred in a London borough ; these were attributed to cockles sent from Leigh-on-Sea and retailed in London. Enquiry showed that similar illness had followed the consumption of crabs and prawns obtained from the same shop on the same day, and the most likely explanation of these events would be that all the foods in question were heavily contaminated with non-specific organisms in the shop, for each kind of food was obtained from a different source.

In 156 instances where reliable histories are available, the average period between the consumption of the food and the onset of symptoms was 16 hours ; the shortest interval was 3 hours and the longest 72. Cases occurring in the same group or family tended to have comparable latent periods, but this was not invariable. Some groups ate a portion of the cockles as soon as bought, and took the remainder home to be shared with other members of the family. When this happened, illnesses reported in the latter, were invariably more serious and prolonged, so that obviously the severity of the symptoms could be correlated with the opportunities for bacterial proliferation.

Detailed symptomatology was available for 84 cases, in most of which vomiting, diarrhoea and griping were present. Diarrhoea was, however, absent in two cases, vomiting in 17 and griping in 15. Most of the illnesses were comparatively trivial and recovery was quick, but a few people, mainly elderly, were more severely affected ; no deaths were reported.

The Cockle Industry

The cockle industry at Leigh-on-Sea is one of the most important of the kind in the country ; the 13 firms engaged in cockling are for the most part, family concerns. Each is based on a " shed " where the cooking is done ; from some a substantial retail trade is carried out. The cockles, which live just below the surface of the estuarine sands are harvested with a short-handled steel rake when the tide allows of access, and are brought to the Leigh Creek, on the side of which the sheds are situated.

In summer cockles are usually cooked immediately on the return from the fishing grounds. Steam is raised in a vertical boiler, and passed through a reducing valve to the cooking pots, in which a vent prevents any significant pressure of steam being built up. After cooking for six minutes the fish are emptied into a rectangular riddle suspended over a 30-gallon tank of fresh water, obtained from the public supply, and the riddle worked backwards and forwards in order to separate the bulk of the shells from the fish, which together with sand and small fragments of shell, fall into the tank.

To facilitate cooling and the removal of debris the fish are then transferred to a series of tanks where they are stirred round in water by a circular movement of a net mounted on a short handle. From the fish brought to the surface in the net, fragments of shell and broken fish are picked out by hand. These operations involve the repeated immersion of the hands and forearms of the operators *and so afford opportunities for post-cooking contamination.*

When freed from extraneous matter the fish are salted. Those destined for distant markets are laid down with a considerable concentration of coarse salt ; they are, in fact, packed in heavy brine. Fish delivered to Billingsgate Market or direct to retailers in Essex and Kent are less heavily salted, while those sold locally are only salted sufficiently to bring out the flavour of the flesh. It is significant that no reports of illness due to heavily-brined cockles were received, and but few cases were attributed to the intermediately salted fish. The main source of trouble appeared to be cockles salted only " to taste ".

Retail distribution in Southend-on-Sea is usually by shops and stalls specialising in shellfish, by hawkers, and from a few fishmongers' shops ; an enormous trade develops at the height of the summer at these shops and stalls, where the fish is arranged in heaps which, displayed in the open air, are repeatedly handled and re-shaped by the assistants, there being thus ample opportunity for contamination to occur.

No cases of food poisoning were attributed to fish sold locally by hawkers, a likely explanation being that their stocks are invariably disposed of before there is time for any serious bacteriological proliferation.

The public health control of the industry is complicated. Most cockles come from " prohibited areas " specified in the Order made by the Port Health Authority of the Port of London, pursuant to the Public Health (Shellfish) Regulations, 1934. The methods of cooking are therefore the concern of the Port Health Authority. Thereafter control is exerciseable by the local authority under the Food and Drugs Act, 1936, Section 13. The events here described called for the closest liaison between the officers concerned, and the writers are indebted to Dr. M. T. Morgan, C.M.G., M.C., Medical Officer of Health of the Port of London, and Mr. Madeley, Port Sanitary Inspector, for their co-operation and assistance.

There are difficulties about investigating food poisoning occurring to a day tripper, who often commits a series of dietetic indiscretions starting from the moment he begins his outing. He may consume beer, ice cream, candy floss, pastry, jellied eels and shellfish but any unpleasant results are almost invariably blamed on the last-named food ; nevertheless an average year produces very few complaints.

It is difficult to find out where the shellfish have been bought ; many of the shops are clustered together, making identification uncertain. Certain families are engaged extensively in this business, so that the same name may be exhibited over shops which have no common source of supply, and the cocklers lend and borrow cooked fish among themselves, so that there may be further confusion about the source of the fish. Inevitably there is a lapse of time before information about food poisoning comes to hand, particularly about patients from other localities, and, when it comes, it is often exasperatingly incomplete and inconclusive.

The administrative problem presented by these events was difficult. The outbreak had three successive phases ; a wave involving three different foods, mainly from one source ; a second wave involving nothing but cockles from sources which were readily identified ; and finally one in which only a small proportion of the cases could be traced back. At the beginning it was impossible to make a hypothesis which would explain all the observed facts, and the conclusions that have been reached rest primarily on negative findings and the rejection of alternative explanations, but subsequent laboratory work has produced significant positive evidence.

There were those who pressed for the prohibition of the sale of shellfish, but it is difficult to understand under what powers this action could have been taken. The Food and Drugs Act, 1938, Section 18, provides that where there are reasonable grounds for suspecting that any food *which has been sampled* is likely to cause food poisoning, a medical officer of health may give interim directions prohibiting its use for human consumption. If later he is satisfied that the food is likely to cause food poisoning, it can, of course, be dealt with like any other unsound food, but if it is not shown to be unsound, the local authority is liable to pay compensation.

In this outbreak there was no one source from which food consistently gave rise to illness, and when illnesses had been notified the stocks in question had all been sold and consumed. Bacteriological examinations produced no recognised pathogens, and all we knew was, that some fish gave high non-specific counts, without any substantial information that this degree of contamination could regularly and consistently cause food poisoning. It seemed clear that the conditions which would have justified action under Section 18 never existed during the outbreak.

The weather conditions during this period were noteworthy. In Southend-on-Sea the month of September was particularly warm, the mean temperature, 65.8°(F.), being 6.1° above the September average, and on September 5th the maximum temperature was 89°, which figure is only recorded on one other day, viz. June 27th. Not only were abnormally high temperatures recorded, but there was a serious drought, so that unusual opportunities for contamination with dust and for the proliferation of organisms undoubtedly existed. The drought affected the condition of the Thames, and it is questionable as to whether there has ever been a period when the contamination of its waters was so high and so sustained.

Summary

An outbreak of gastro-enteritis associated with the consumption of shellfish continued for about four weeks during abnormally hot weather. It is attributed to the presence, in the processed fish, of a large number of non-specific organisms. These did not survive the cooking process, which was effective, but were introduced in the subsequent handling of the product. This contamination would not, in ordinary conditions of weather and temperature, have resulted in growths of such magnitude as to cause illness, even in susceptible persons, but during a period of sustained abnormally high temperatures bacterial proliferation was stimulated to such an extent as to do so.

As Dr. Pilsworth shows later, the cooking process when properly carried out invariably produces a sterile fish, which can be rapidly contaminated by the subsequent processing: good methods can reduce this to negligible proportions. The mean daily temperature is a most significant factor in determining the bacterial content of the fish, but his studies show that the retailer, too, has a considerable responsibility for the safety of the product. Samples taken from different retailers who are supplied with the same batch of fish sometimes show very different results, but with good technique a retailer can maintain his product in the condition in which he receives it from his supplier.

Much work in the two subsequent seasons has confirmed the view formed originally about this outbreak. It has been followed by intensive attention to techniques throughout the trade, the encouragement of good methods, and extended use of refrigeration. These factors have accomplished much,

but more work is required with the object of working out a two-stage cooking process which will be acceptable commercially. The ideal arrangement would be, to (*a*) cook the fish sufficiently to facilitate removal from the shell, (*b*) wash to separate all extraneous matter (*c*) cook to sterilise.

Grateful acknowledgments are made to Dr. J. C. Preston, deputy M.O.H. who, in the absence of the Medical Officer of Health, was in charge during the greater part of the outbreak, to Mr. R. A. Drake, B.E.M., chief sanitary inspector, to whose careful and unrelenting enquiries much of our information is due, and to Dr. M. T. Morgan, C.M.G., M.C., Medical Officer, London Port Health Authority, and Mr. Madely one of his inspectors.

BACTERIOLOGICAL STUDIES ON COOKED SHELLFISH

R. Pilsworth, M.D., Dip.Bact. Director, Southend-on-Sea Laboratory
(Public Health Laboratory Service)

Much work has been carried out on the bacteriology of shellfish such as oysters which are eaten uncooked, but those types of shellfish such as cockles, which are often sold ready cooked, present different bacteriological problems and require different methods of examination. Briefly, the reasons for these differences are as follows :—

1. Shellfish normally eaten raw receive their principal contamination from the water in which they lie. Consequently the most usual pathogenic contaminants in this case are members of the *Salmonella* group, together with " Indicator " organisms such as faecal *Bact. coli.* Cooked shellfish, however, contain few endogenous organisms and are chiefly contaminated directly from human sources in the period between cooking and consumption. Consequently the fish may be contaminated by a large variety of pathogens and " borderline pathogens " of human origin.

2. Fish that remain in their shells up to the time of consumption constitute a series of distinct units. Contamination does not easily spread from one individual to another ; otherwise the bacteriological purity of such shellfish could not be reported by the " percentage clean " method. On the other hand, cooked shellfish out of their shells form a homogeneous mass through which bacterial contamination may spread rapidly and uniformly, so that the bacterial flora of one individual is usually representative of that of the whole mass.

3. Fish eaten alive retain, up to the point of consumption, a resistance to bacterial invasion characteristic of the living organisms. Cooked shellfish provide an inert and nutritious medium for the growth of bacteria.

Cooked shellfish, then, resemble " made-up " foods, such as meat pies, custards, trifle, etc., with two important differences ; the conditions under which the fish are processed and stored can fall short of those which would be ideal in the kitchen, bakery or food shop and in addition there is always the risk of the cooked fish becoming recontaminated by uncooked shellfish awaiting treatment by the processor. The nature of the material and the high water content will allow rapid bacterial multiplication under suitable conditions.

The process of contamination to which cooked shellfish are exposed is continuous, from their emergence from the cooking pot up to the time of consumption, so that their bacterial population rises progressively at a rate varying with the temperature and the degree of hygiene with which they are handled.

During the Southend 1949 outbreak of food poisoning no specific pathogens were isolated from the shellfish on sale at that time or from the excreta of affected persons. During the following two years the examination of over a thousand representative samples failed to yield organisms of the *Salmonella* group or *Staph. pyogenes.* At the time of the outbreak attempts to implicate chemical substances were equally unsuccessful, and at first sight the causes of this outbreak would seem obscure. However, the number of types of organism capable of causing food poisoning has, over the last ten years or so, been recognized as being larger than was originally believed, and it has been realized that certain species, particularly the

" borderline pathogens " such as *Proteus* species, viridans streptococci, etc., may give rise to gastrointestinal symptoms when ingested in large numbers. *Proteus vulgaris* and *Proteus morgani* have long been suspected, and recent work suggest that *Bact. coli* and *Bact. aerogenes* (Jordan and Burrows 1935), paracolon bacilli (Edwards 1943, Stone 1944), anaerobic sporebearers (Duncan 1944) and α haemolytic streptococci (Dack 1943, Moore 1948) may all play a part in the genesis of food poisoning. In considering this varied array of different species it would be surprising if, under appropriate conditions one or other of them did not, at some time, find opportunities to multiply abundantly in cooked shellfish. Though the occurrence of an outbreak of food poisoning due to an organism of the *Salmonella* group will depend on the moderately rare coincidence of the presence of an excreter of this organism together with such conditions of hygiene, temperature and incubation period as are necessary for bacterial contamination of and proliferation in the food in question, in " non-specific " food poisoning, on the other hand, no such coincidence is necessary, as the organisms responsible are almost ubiquitious, particularly in the human environment, and the more general factors are the only conditions required. Again, an outbreak due to a specific organism will arise from one particular focal point, to which it may, with good fortune, often be traced back. The ubiquity of the " non-specific " organisms referred to will make it possible for an outbreak to commence at several points simultaneously if sufficiently high temperatures and low standards of hygiene operate uniformly at each point. This is what apparently occurred in the Southend outbreak of 1949. Under certain conditions, then, it would seem that the total bacteriological activity present in a given food might be of as much importance as the presence of a specific pathogenic organism. Consequently attempts have been made to include indirect studies of the total bacterial populations of cooked shellfish, in addition to the isolation of specific pathogens.

Methods of Examination

1. " Routine examination." During the emergency of the outbreak and in the following months a simple plating method was used to assess roughly the aerobic bacterial population. A cockle was wiped over the surface of half a 5 per cent. horse blood agar plate ; afterwards it was bisected with sterile scissors and the cut surface wiped over the other half of the plate. After incubation at 37° C. for 24 hours the plate was examined and a report made in arbitrary terms varying from " Very poor " to " Very good " according to the paucity of bacterial growth. It was felt that this method might not be sufficiently quantitative for comparing the bacterial quality of different batches of shellfish, so that the following method was devised and used, at first in parallel with this method.

2. *The Methylene Blue Test.* Cooked shellfish resemble milk and ice-cream in that they are consumed without further treatment, often some time after the original heat treatment, and an attempt has therefore been made to modify the methylene blue test, as used in testing ice-cream, to suit the requirements of cooked shellfish. Details and results of this method, with justification for its employment will appear elsewhere, but briefly the test consists in incubating a known volume of extract of macerated cockle in $\frac{1}{4}$ strength Ringer's solution, together with sterile milk as a substrate, and methylene blue. With producer samples the tubes are transferred to a waterbath at 37° C. after overnight pre-incubation at 20° C. and re-examined for dye reduction after two hours. Certain arbitrary standards, which will be discussed later, were based on the findings for the year 1950, a higher level being expected from producer samples than from retailers.

3. *Bact. coli* isolation. Although as a routine all samples are examined for the presence of *Staph. pyogenes, Salmonella* organisms and *Cl. welchii*, the only other test of interest in connection with this work is the isolation of *Bact. coli*. A suspension of macerated shellfish as used in the methylene blue test, equivalent to one-third of the total volume of the fish, is inoculated into a tube of single strength liquid MacConkey's medium. Tubes showing acid and gas after 48 hours at 37° C. are subcultured into tubes of the same medium held at 44° C. for 24 hours. Cultures showing acid and gas at the end of the period are plated on to MacConkey's agar and suspicious colonies examined by the IMVIC series of reactions to confirm the identity of faecal *Bact. coli*.

The method of Clegg and Sherwood (Clegg and Sherwood 1947) was used occasionally when a more accurate estimation of faecal coli was required; but it was felt that the mere presence of faecal coli in a food that had, at one point, been sterilized, was sufficient evidence of undue contamination, and that, except for special purposes, this method was an unnecessary refinement.

Stages of Possible Contamination :

Studies were carried out on cooked shellfish at various stages during processing to find out where the most important degree of contamination occurred. Cockles were examined:

1. Before cooking.
2. Immediately after cooking.
3. After riddling.
4. After washing.
5. While held by the retailer.

1. *Raw shellfish*

To find out the degree of contamination normally encountered a number of uncooked shellfish was examined using the technique of Clegg and Sherwood for the enumeration of faecal *Bact. coli*. In a series of twelve counts the numbers of faecal *Bact. coli* per cockle ranged from 2 to 52 with a mean of 12.

2. *Immediately after cooking*

TABLE I

The effect of steaming cockles heavily impregnated with faecal *Bact. coli*

Experiment	*Bact. coli* per ml. suspension	*Bact. coli* per cockle before steaming	*Bact. coli* per cockle after 1½ mins. steaming	*Bact. coli* per cockle after 6 mins. steaming
1	200,000	47,000	0	0
2	215,000	21,000	0	0
3	150,000	13,000	0	0

To test the effectiveness of the cooking process, batches of live cockles were laid overnight in artificial seawater heavily impregnated with faecal *Bact. coli*. Next morning counts were performed on the water and on the shellfish, both live and after exposure to free steam for periods of 1½ and 6 minutes. Table I shows that the faecal *Bact. coli* counts per cockle were much higher than those normally encountered, but the steaming process reduced them to zero after only 1½ minutes.

Table II

Bacterial findings on batches of cockles sampled at two stages of processing

	Routine Assessment		Methylene Blue reduction	
Shed	From steamer	After last wash	From steamer	After last wash
A	Very good ...	Poor	Over 4 hours ...	P
A	Very good ...	Poor	Over 4 hours ...	P
B	Very good ...	Good	Over 4 hours ...	3 hours
B*	Very good ...	Good	Over 4 hours ...	Over 4 hours
C	Very good ...	Poor	Over 4 hours ...	P
C	Very good ...	Good	Over 4 hours ...	3 hours
E	Very good ...	Good	Over 4 hours ...	4 hours
E	Very good ...	Fair	Over 4 hours ...	2½ hours
G	Very good ...	Fair	Over 4 hours ...	1 hour
J	Very good ...	Very poor	Over 4 hours ...	P
J	Very good ...	Poor	Over 4 hours ...	P
L	Very good ...	Fair	Over 4 hours ...	2½ hours
L	Very good ...	Very poor	Over 4 hours ...	P
N	Very good ...	Poor	Over 4 hours ...	1 hour
P	Very good ...	Fair	Over 4 hours ...	1 hour
Q	Very good ...	—	Over 4 hours ...	—

P = methylene blue reduced during the overnight pre-incubation period at 20°C.

* It was stated that "special precautions" had been taken by the producer, as he was expecting a sample to be taken.

Table III

Results of Methylene Blue Tests performed on 87 batches of cockles samples at two stages of processing

Methylene Blue Reduced	Immediately after steaming	After final washing
	Per cent.	Per cent.
0–1 hours	—	27·5
1½–2½ hours	—	15·0
3–4 hours	—	30·0
Over 4 hours	100	27·5

Table IV

Results of Methylene Blue Test performed on 4 batches of cockles at several stages of processing

(Figures refer to methylene blue reduction time in hours)

	1	2	3	4
Raw shellfish	3½	1½	0	—
After steaming	Over 4½	Over 4½	Over 4½	Over 4½
From riddling tank	Over 4½	0	1	Over 4½
2nd wash	Over 4½	0	1	Over 4½
3rd wash	2	½	1	—
4th wash	1½	0	1	—
Final wash	1½	½	1	3

When shellfish were sampled at various stages of commercial processing it was found that fish taken directly from the steaming-pots were practically free from non-sporing organisms and did not decolourize methylene blue rapidly (Tables II, III, IV).

3. *After riddling*

At one time it was suggested that cooked shellfish might be exposed to severe contamination derived from organisms embedded in the mud covering the shells. These organisms might be protected by the mud and escape death during the steaming process. The violent agitation to which the shells are subjected during the riddling process normally chips off pieces of mud and flakes of shell which might release any such "protected" organisms into the cockle meat. However, repeated examination of the ground-up shells of cockles that had been exposed to free steam for $1\frac{1}{2}$ minutes failed to show any such release of organisms.

A more likely source of contamination at this stage is from the riddle itself, which consists of a wire grid of about $\frac{2}{3}''$ mesh and is virtually impossible to sterilize efficiently.

4. *After washing*

The purpose of this process is to remove sand and pieces of broken shell from the cockle meat and to induce rapid cooling and is not directed towards hygienic ends, as borne out by Table II, III, and IV. The open nature of the tanks used and the mode of operation provide ample opportunity for contamination of the fish by organisms of human origin. Solutions to the problem of washing the fish without introducing any appreciable degree of contamination are simple in theory, but difficult in practice and may involve either large amounts of running water at this stage or the employment of a "Pasteurising" process at a later stage.

5. *While held by the retailer*

TABLE V

Methylene Blue reduction and *Bact. coli* isolation in 65 producer samples during the year 1950

	Reducing MB in 1 hour or less	Presumptive *Bact. coli* present	Faecal *Bact. coli* present
Producer samples ...	15 (23 per cent.)	6 (9 per cent.)	0
Retail Samples ...	33 (66 per cent.)	26 (52 per cent.)	8 (16 per cent.)

Table V shows the results obtained for 65 producer samples and 50 retail samples during the year 1950. Of the former only 23 per cent. reduced methylene blue in one hour or less, 9 per cent. contained presumptive *Bact. coli*, while faecal *Bact. coli* were consistently absent. Of the retail samples, however, 66 per cent. reduced methylene blue within one hour or less 52 per cent. contained presumptive *Bact. coli* and 16 per cent. contained faecal *Bact. coli*. The appearance of faecal *Bact. coli* in only retail samples suggested that a considerable degree of contamination might occur on the premises of the retailer, but the findings for the year 1951 (Table VI) were slightly different. During this period 680 sample were examined—450 producer and 230 retail samples.

TABLE VI

Methylene Blue Results and *Bact. coli* isolation in 450 producer samples and 230 retail samples during the year 1951

	Failed appropriate M.B. test	Presumptive *Bact. coli* present	Faecal *Bact. coli* present
	Per cent.	Per cent.	Per cent.
Producer	23·2	17·8	2·9
Retailer	38·8	29·1	6·9

Faecal *Bact. coli* were isolated from 2·9 per cent. of producer samples as against 6·9 per cent. of the retail samples. These figures are probably more representative and suggest that faecal contamination is not confined to the retailer; it is possible that faecal *Bact. coli,* introduced in small numbers by the producer, are not easily detectable until after a period of incubation so that a higher proportion of retail samples will be found to contain them. At the same time there is ample opportunity for contamination in the smaller booths and stalls, although the larger concerns possess cold rooms or refrigerators for storage purposes and show a high standard of hygiene in handling the fish.

It is difficult to assess accurately what extra amount of contamination results from retail handling, considering the variable condition of the fish as it arrives from the producer. The retailer can never supply a sample of a higher bacterial standard than that of the fish supplied by the producer and it is difficult for him even to maintain this standard if he has to keep it for any length of time in the absence of refrigeration.

Because the retailer is necessarily handicapped in this way the criteria governing the assessment of the methylene blue test were modified so as to be less exacting for retail samples. These criteria were adjusted to the figures obtained during 1950, so that at least 50 per cent. of all producer samples might be expected not to reduce methylene blue within two hours of incubation at 37° C., and at least 50 per cent. of all retail samples should not reduce the dye during the overnight pre-incubation period at 20° C. It was emphasized that these criteria were provisional and arbitrary and that any opinion as to the standard of purity of the product of any one producer or retailer should be based on a consideration of a number of samples, and of these at least 50 per cent. should pass the respective test. During the year 1951, 76·8 per cent. of producer samples and 61·2 per cent. of retail samples came up to their respective standards (Table VI). The year 1951 was, on the whole, not notable for any period of sustained high temperature and this high proportion of "Satisfactory" samples is not surprising. It should be noted also that the criteria mentioned above were considered in relation to other findings; for instance it was considered that even if the methylene blue test was satisfactory, the presence of faecal *Bact. coli* in a sample should render it "unsatisfactory." On no occasion have organisms of the *Salmonella* group or *Staph. pyogenes* been isolated from cooked shellfish.

Summary

1. Cooked shellfish present bacteriological problems peculiar to themselves and differ from shellfish normally eaten uncooked in the methods by which they should be examined.

2. The bacteriology of cockles at various stages in processing has been studied using a modified methylene blue test, a simple plating method and tests for presumptive and faecal *Bact. coli.*

3. The steaming process appears to destroy all non-sporing organisms derived from sea water.

4. The process of riddling does not appear to release organisms that have been protected by dried mud from the action of the steam, because no such protection can be demonstrated.

5. During the washing process a considerable degree of contamination appears to take place, mainly from human sources.

6. Although the bacterial population may increase while shellfish are held by the retailer, this is probably more a function of time and temperature rather than of contamination derived from the retailer. Ample opportunities for contamination do, however, occur in many retail concerns.

7. The Southend outbreak of 1949, ascribed to cockles, would appear to be characteristic of the type caused by " non-specific " bacterial agents and associated with high temperature and poor hygiene, rather than the type due to a specific organism associated with the presence of a human excreter.

References

Clegg, L. F. L. and Sherwood, H. P. (1947) *J. Hyg. Camb.* **45**, 504.

Dack, G. M. (1943) " Food Poisoning " Univ. Chicago Press, Chicago, Ill.

Duncan, J. T. (1944) *This Bulletin*, **3**, 61, (April).

Edwards, J. L. (1943) *Ibid.*, **2**, 84, (September).

Jordan, E. O. and Burrows, W. (1935) *J. infect. dis.*, **57**, 121. (Quoted in " Principles of Bacteriology " Topley and Wilson, Arnold, London, 1946).

Moore, B. (1948) *Bull. Hygiene* **23**, 883.

Stone, D. M. (1944) *This Bulletin*, **3**, 58, (March).

OPHTHALMIA NEONATORUM

A Review of the Prophylactic methods at present in use

Rachel A. Elliott, M.D., D.P.H., Ministry of Health

The need for continuation of the traditional methods of prophylaxis was raised a short time ago and as a result an enquiry was made into the present practice obtaining in both institutional and domiciliary midwifery.

Replies were received from, or on behalf of, approximately 250 maternity hospitals and from the medical officers of health of 148 local health authorities.

Some difficulty has been experienced in tabulating the information given as practice was found to vary considerably even within a given unit or area, e.g., in several of the larger counties varying methods were used in their several Divisions and many authorities reported that the midwives followed the method of their various training schools and so there was not necessarily uniformity of practice. Figures, therefore, were not always strictly comparable but they give a general picture of the position.

Methods in Use

	No chemical prophylactics	Silver preparations*	Other chemical prophylactics†
Hospitals and Maternity Units	139	43	68
Local Health Authorities ...	74	86	34

* Including silver nitrate, collosol argentum, protargol, argyrol, etc.

† Including albucid, penicillin, boracic lotion, etc.

In considerably more than 50 per cent. of the maternity units the routine use of prophylactics has been abandoned, while in nearly 40 per cent. of local health authorities areas no chemicals are in use as a routine measure.

It should be added, however, that in the majority of cases, whether institutional or domiciliary, it was reported that, where no prophylactics are in regular use, chemicals are employed for emergency or unbooked cases and where there is any reason to suspect the presence of infection in the mother.

Results where no chemical prophylactics are used

In very few of the replies received was a distinction drawn between the effects of change in methods of prophylaxis on frank ophthalmia neonatorum or on sticky eyes, so that, for the purpose of this review, both are grouped together.

Where no prophylaxis, other than cleansing with sterile water, dry swabbing or saline swabbing is undertaken, the results may be tabulated as follows:—

	No change in incidence	Decreased incidence	Increased incidence	No comparative figures	Doubtful or no reply
Hospitals 139	79	40	2	6	12
L.H.As. 74	48	25	1	—	—

These figures indicate that, in the absence of the routine use of chemical prophylactics, no change or a definite decrease in the incidence of infection has been noted.

Results where recent changes have been made

In 62 hospitals where there have been recent changes in routine 48 are not now using chemical prophylactics of any kind. In 20 of these no change in incidence has occurred while 25 report a decrease and only 3 an increased incidence of infection.

In a further 14 maternity units where changes have been made recently in the type of chemical prophylactic used, e.g. from the various silver preparations to penicillin, albucid, etc., or to a different silver preparation, in 4 there has been no change in incidence while in 9 a decrease is reported and in only 1 were the results doubtful.

Similarly, 32 local health authorities reported changes in routine prophylaxis during recent years. Of these, 19 are not now using any chemicals and, as a result, in only one was an initial increased incidence reported and this decreased later.

In the remaining 13, changes were made in the chemical prophylactics used as a routine but the results show that the incidence was unchanged or decreased in 9, increased in 1 and doubtful in 3. It does not, therefore, appear that great importance can be attached to the type of chemical prophylaxis employed.

Various trials have been, or are being, conducted both by maternity hospitals and certain local health authorities. These show that, on the whole, results are as good, if not better where no prophylactics are used as when the various chemicals are employed. The view has been expressed that there is no disadvantage in omitting silver nitrate and it is widely held that simple cleansing of the eyelids is as effective as any other form of prophylaxis so far described, though, as will be seen from the results given above, not all subscribe to this view.

In commenting on results, several obstetricians and medical officers of health made some observations which have a considerable bearing on the subject. These may be summarised as follows:—

(1) As ophthalmia neonatorum and sticky eyes appear several days after delivery the infection is almost certainly not acquired during delivery.

(2) Many of the older prophylactic drugs produce chemical irritation of the eyes and do more harm than good.

(3) The manner in which the eyes are handled at birth is of considerable importance in the avoidance of trauma and subsequent infection.

(4) Strict bacteriological control of staff and the exclusion of all carriers of virulent staphylococcus aureus are necessary to reduce the incidence.

(5) The condition of sticky eyes appears to be solely one of cross infection either from infected carriers in the staff or in the nursery, as shown by phage types.

(6) It is important to move the baby away from the near vicinity of the bed during bed-making in order to protect the eyes from blanket dust.

(7) Medical aid is likely to be summoned more promptly when no chemical prophylactics are used. In the past there might have been a temptation for some midwives to fail to notify sticky eyes on the grounds that the condition was a reaction after the use of silver nitrate.

(8) Much seems to depend on the midwife's conception of the term "sticky eye" so that it is necessary to exercise care in forming any conclusions based on the reports of sticky eyes.

Summary

The results of an enquiry into the present practice with regard to prophylaxis of ophthalmia neonatorum have been collated. These show that in domiciliary and institutional midwifery 40 per cent. of local health authorities and 50 per cent. of hospitals have abandoned the use of chemical prophylactics, that, on the whole, there is no appreciable change in the incidence of ophthalmia neonatorum or sticky eyes as a result but that in a considerable number the incidence is lowered, while in only a very small percentage is it reported to be increased.

Several authorities and hospitals have changed from the use of one chemical to another without any appreciable alteration in results and mainly, but not altogether, the trend has been away from the silver preparations.

Several hospital have conducted or are conducting trials but the results on the whole have not been conclusive though it would appear that the results are as satisfactory without the use of chemical prophylactics as when these are employed.

A number of relevant comments on the subject have been added.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, MAY, 1952

(Issued from the General Register Office, Somerset House, W.C.2.)

	May 3	May 10	May 17	May 24	May 31	Average weekly figures for May, 1951
Scarlet Fever	877	1,033	1,088	972	915	996
Whooping Cough	3,116	2,993	2,905	2,785	2,595	3,063
Diphtheria	21	16	27	25	24	39
Measles, excluding Rubella ...	4,743	4,494	5,749	6,246	6,616	18,968
Acute Pneumonia	582	491	433	430	380	565
Meningococcal Infection ...	39	40	43	42	31	34
Acute Poliomyelitis (Paralytic)	15	15	25	18	26	16
,, ,, (Non-Paralytic)	9	6	12	19	18	8
Ophthalmia Neonatorum ...	36	36	36	44	39	31
Puerperal Pyrexia and Puerperal Sepsis	254	267	245	255	225	83
Dysentery	241	367	328	269	276	781
Paratyphoid	10	5	22	53	45	10
Typhoid	5	3	1	1	3	5
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1

Sheffield Laboratory

On the resignation of Dr. L. Gordon Cook to take up a post in New Zealand, Dr. E. H. Gillespie has been appointed Director of the Public Health Laboratory at Sheffield from the 12th May, 1952.

British Standards Institution

Readers of the Bulletin may be interested to know that the Institution has recently published a new British Standard (B.S.1752 : 1952) for sintered disk filters ranging in maximum pore size from 500μ to 2μ.

STAPHYLOCOCCUS AUREUS IN A SLAUGHTERHOUSE

G. B. Ludlam, M.B., Ch.B., D.T.M. & H., D.L.O., Public Health Laboratory Service, Nottingham

In the course of an investigation of carcasses in a slaughterhouse for the presence of salmonellae, it was decided to inquire also into the occurrence of *Staphylococcus aureus*. This organism is an important cause of food poisoning and no similar investigation appears to have been recorded.

Methods

The slaughterhouse (No. 1) in which most of the work was done was a large municipal building of good design where the routine practices were probably at least as satisfactory as in other similar slaughterhouses.

The carcasses were swabbed on the same day as the killing, usually within one or two hours of slaughter. The slaughterers' implements and other slaughterhouse equipment were swabbed either during use or during the lunch hour when the gear was hanging up. A note was kept of the visible dirtiness of the aprons, clothes, etc., but no relationship was found between the naked eye appearances and the isolation of *Staph. aureus*. The carcass swabs were obtained in a total of 15 visits to the slaughterhouse. All the swabbing of the equipment was carried out at two visits within a short period of each other.

A short investigation of a second small slaughterhouse (No. 2) was carried out in two visits within a few days of one another. The same techniques were used as for the first slaughterhouse.

Swabbing technique. The swab used was about the size of a half-walnut and was secured on the wooden applicator by a little paste before sterilizing. The swabs were moistened with sterile 1/4 strength Ringer solution before use.

The beasts were skinned immediately after killing, and the carcasses slung on an overhead runner. The carcasses were swabbed while hanging in this way, on the subcutaneous fat or fascia of the saddle area of the back. In the earlier part of the work swabs were taken also from the peritoneal surfaces ; *Staph. aureus* was isolated as frequently as from the subcutaneous surface. An area on the meat of about 9-12 inches diameter was vigorously rubbed and the swab then broken off into a 1 oz. screw-cap bottle either empty or containing about 10 ml. of 10 per cent. salt meat broth (Maitland and Martyn, 1948). Equipment was swabbed as far as possible in the same way as the carcasses.

Cultural technique. The swabs were cultured in salt meat broth for 48 hours without preliminary plating on a solid medium, as it was found at an early stage that direct plating gave distinctly fewer isolations. The salt meat broth cultures of the first 79 carcass swabs and swabs of other materials were plated on 6·5 per cent. salt agar and a tellurite agar (Ludlam, 1949). The tellurite agar gave rather better results than the salt agar but more isolations were obtained by using both media. The salt broth cultures from the next 40 swabs were also plated out on a phenolphthalein phosphate agar medium (Barber and Kuper, 1951). This gave more isolations than the other two solid media and for the remainder of the carcasses swabbed in this slaughterhouse this medium alone was used. The sodium phenolphthalein phosphate used was the remainder of a 10 per cent. solution kindly presented by Miss Joyce Cranfield of Edinburgh University Bacteriology Department and obtained by her originally in 1949 from Hopkin and Williams, Ltd. A supply of the solid sodium salt obtained later in 1951 gave very unsatisfactory results, liberating relatively little phenolphthalein and giving poor distinction between *Staph. aureus* and *Staph. albus.* A sample received from Dr. Mary Barber on the other hand gave very ready liberation of phenolphthalein but distinction was again poor.

In the work with the second slaughterhouse the same tellurite medium was used but at a neutral pH as batches of the original medium were proving very inhibitory at this time. A phenolphthalein phosphate agar made with the unsatisfactory sample of the reagent was also used but better results were obtained with the tellurite medium.

After 24 and 48 hours' incubation on these various solid media the cultures were examined, suspected colonies were picked off on to nutrient agar and the resultant growth tested for coagulase production. In order to obtain a fair comparison between them not more than three colonies were examined from each medium.

Phage typing. Random colonies of *Staph. aureus* from a number of carcasses and from other materials in the slaughterhouse were phage-typed by the Staphylococcus Reference Laboratory.

Results

It can be seen from Tables 1 and 2 that *Staph. aureus* was widespread in two slaughterhouses—the first a large, modern well-run place and the second small and in rather unsatisfactory premises. In the first slaughterhouse *Staph. aureus* was isolated from every batch of swabs examined, the incidence of positive swabs varying from 21 out of 22 to 4 out of 12. The total incidence was 113 positive carcasses out of 178 examined (63·5 per cent.). Culture of objects coming into regular contact with the carcass at the time of skinning also yielded *Staph. aureus* not uncommonly. All materials cultured gave a heavy growth of organisms, predominantly of a coccal type and often consisting partly of pigmented coagulase-negative staphylococci, sometimes indistinguishable on colonial characters from *Staph. aureus.* Because of this, identification of colonies of *Staph. aureus* was not easy. The proportion of positive swabs obtained was found to vary with the type of solid medium used and the best results were obtained by using more than one medium. Probably with greater diligence and better methods of isolation a still higher proportion of positive results could have been obtained.

TABLE 1

Staph. aureus in Slaughterhouse 1

Source of swabs	Number swabbed	Number positive for *Staph. aureus*
Carcasses	178	113 (63·5 per cent.)
Slaughterers' aprons	13	5
Hatchets	5	2
Knives	7	3
Saw	1	1
Wiping cloths	3	3
Sweeping brush	1	0
Various parts of floor	7	2

TABLE 2

Staph. aureus in Slaughterhouse 2

Source of swabs	Number swabbed	Number positive for *Staph. aureus*
Carcasses	11	8
Knives	8	7
Saw	1	1
Mechanical saw	1	0
Hatchet	1	1
Nasal swabs from workers	5	1

Although it seemed probable that the meat was contaminated at the time the carcasses were skinned, it was possible that contamination might have arisen from the hands and clothes of workers at a rather later stage. Tables 3 and 4 show that in both slaughterhouses the organisms isolated from the meat were commonly of the same phage types as those found on the equipment used in dressing the carcasses. A positive nasal swab from a worker in the second slaughterhouse yielded organisms of a different type from those isolated from the man's surroundings.

TABLE 3

Phage-typing of Strains of Staph. aureus from Slaughterhouse 1

Source of Strains	Number of Strains Reacting with Phages				No reaction with phages
	6 and 53	One or more of 7, 52A, 54	42B	52B	
Carcasses	4	6	0	2	8
Tools, cloths, etc. ...	5	3	1	1	3

TABLE 4

Phage-typing of Strains of Staph. aureus isolated in Slaughterhouse 2

Source of strains	Number of strains reacting with phages						No reaction with phages
	7/47C/54	7/54	29	70	75A	78	
Carcasses	1	2	0	0	0	1	0
Tools	1	0	2	0	1	2	1
Nose of slaughterer	0	0	0	1	0	0	0

Where direct plating was carried out it proved much inferior to preliminary culture of the swab in salt meat broth. For example, in the second slaughterhouse 4 out of 11 carcasses and 1 out of 8 knives were positive by direct plating as against 8 out of 11 and 7 out of 8 respectively by enrichment in salt meat broth. These results suggest that usually *Staph. aureus* was only present in small numbers. Direct plating always yielded a very heavy growth of predominantly coagulase-negative cocci among which scanty colonies of *Staph. aureus* were liable to be lost.

Discussion

Since *Staph. aureus* is commonly found in the air and dust of occupied rooms it should perhaps not be surprising that it was found widespread in a slaughterhouse. On the other hand, the floors of both slaughterhouses were frequently hosed down and the atmosphere was damp and cool. There was therefore little opportunity for contamination of the air with dust although a certain amount could have been liberated from the clothing of workers. The presence of *Staph. aureus* on the knives and wiping cloths, and the phage-typing results, suggest strongly that certain strains of staphylococci were widespread throughout the slaughterhouse and that these reached the meat by contact with the knives and cloths at the time the carcasses were dressed. If this is so, it seems possible that even the simple measures for sterilization of knives and cloths recommended by the Interdepartmental Committee on Meat Inspection (Report, 1951) might be sufficient to reduce materially the staphylococcal flora of the slaughterhouse.

It seems improbable that the contamination of meat at the time of dressing the carcasses is normally of much hygienic importance. The number of *Staph. aureus* deposited on the meat appears to be small, and so long as the meat is kept cool multiplication may be expected to be slight. Where the meat is sold to the public within a few days and then cooked within a day or two the risk should be negligible. Where the meat is made up in factories the risk may be greater if conditions in the factory are not satisfactory (Report, 1950). These results mean at least that the meat is one route whereby *Staph. aureus* is continually reaching the factory however perfect the conditions in the factory may be. In searching for the source of pathogenic staphylococci in a meat factory one first thinks of nasal carriers. Where phage-typing can exclude these as the source of a particular phage type it is to be remembered that the type in question may have originated in the slaughterhouse.

Summary

Staph. aureus was isolated from 113 out of 178 carcasses swabbed in a large and good quality slaughterhouse. The organism was widespread on the aprons and knives and other implements of the slaughterers. Phage-typing showed that the common types in the meat were also the common types on the implements.

A brief investigation of a small slaughterhouse revealed similar conditions. *Staph. aureus* was isolated from the nose of only one of the five men dealing with the carcasses and the phage type was different from the phage types found on the tools and the meat.

These findings suggest that the possibility of slaughterhouse contamination should be borne in mind when attempting to determine the source of staphylococci found in cases of staphylococcal food poisoning due to meat, especially made-up meat products.

I am greatly indebted to Dr. R. E. O. Williams for the phage-typing results and to Dr. Mary Barber for a sample of sodium phenolphthalein phosphate.

References

Barber, Mary and Kuper, S.W.A. (1951) *J. Path. Bact.*, **63**, 65.

Ludlam, G. B. (1949) *This Bulletin*, **8**, 15.

Maitland, H. B. and Martyn, G. (1948) *J. Path. Bact.*, **60**, 553.

Report (1950) Manufactured Meat Products Working Party, Ministry of Food, p. 25.

Report (1951) Interdepartmental Committee on Meat Inspection, Ministry of Food, p. 53.

COPYING OF DOCUMENTS BY REFLEX PRINTING

B. R. Sandiford, M.D., and E. T. J. Chelton, A.I.M.L.T.,
Public Health Laboratory, Birmingham

Although the reflex printing process is not new, some workers may find it useful to have a description of a simple arrangement for the copying of articles, by which a black print on white paper is obtained. The cost per print for a laboratory doing this work only occasionally is about 3d., a price which compares favourably with that usually charged by commercial firms.

Materials required

1. Minimum of two, preferably three photographic dishes, of a size equal to largest prints to be made. Few of the common journals are larger than 10 by 8 inches.

2. Two sheets of plate glass, 12 by 10 inches approximately.

3. 100-watt bulb.

4. Any metol-quinol photographic developer giving good contrast.

5. Hypo fixing solution or, for rapid results, "AMFIX" (May and Baker).

6. Reflex copying paper.

The paper is a bromide paper of low sensitivity (obtainable from Ilford, Ltd. and Kodak, Ltd.). It may be handled in subdued artificial light such as the indirect light from a 15-watt bulb some 6 feet or more away from the working bench. A safelight is not necessary.

Method

1. The article to be copied is placed on one piece of plate glass and a sheet of reflex copying paper is laid on top of the article, emulsion side to print. The second piece of plate glass is laid on top of the copying paper, and hand or mechanical pressure applied to ensure perfect register, without which poor prints will be obtained.

2. Light from the 100-watt bulb is passed from above, *i.e.* through the back of the reflex copying paper. The exposure must be found by trial and will depend on the distance the lamp is placed from the paper. Once the exposure has been determined for a certain distance between light and subject, it will be found fairly constant for different articles.

3. Remove the paper and immerse in developing solution so that the whole of the surface is covered at once. This is important; a wetting agent is useful. Develop by inspection until good blacks and clean whites are observed.

4. Rapidly rinse in water and fix for 15 minutes in Hypo fixing solution (or for a much shorter period in "AMFIX").

5. Wash and dry the paper negative.

6. From the negative prepare a contact print. To do this lay a sheet of reflex paper on the plate glass, emulsion side up, and then place the negative on top, emulsion side down (*i.e.* emulsion to emulsion). Cover with the second piece of plate glass, apply pressure and expose to light through the back of the negative. The exposure necessary will be about double that which was required for the negative.

7. Process as for negative.

Summary

A description is given of the reflex printing process by which documents may be cheaply and quickly copied in the laboratory.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The May Bulletin was issued on 28th May.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(15816) Wt. 8221—7283 3,300 6/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

JULY 1952 Vol. 11

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1.

Editor, Section I

From July 1st, 1952, Dr. J. Balfour Kirk, C.M.G., F.R.C.P., will be the Editor of Section I of this Bulletin, following the retirement of Dr. Glover.

DISTRIBUTION OF POLIOMYELITIS BY SEX, AGE, AND GEOGRAPHICAL AREA

W. P. D. Logan, M.D., Ph.D., B.Sc., D.P.H., Chief Medical Statistician, General Register Office

Although several localised outbreaks of poliomyelitis occurred in England and Wales during the first decade of this century, and a few slightly earlier (Pasteur, 1897 ; Buzzard, 1898), the official statistical history of the disease in this country did not begin until 1911 when poliomyelitis was first shown as a separate cause of death in the national mortality statistics. Poliomyelitis had been designated a notifiable infectious disease in a number of local areas in 1911, and in September 1912 it was made compulsorily notifiable throughout the country. Polioencephalitis became separately notifiable in 1919 and continued so until the beginning of 1950 when it ceased to be separately distinguished for notification purposes from poliomyelitis.

Between 1913 and 1946 a few hundred cases of poliomyelitis, including polioencephalitis, were reported each year, with 1926 and 1938 outstanding in having twice as many cases as usual and therefore looked on at the time as epidemic years. Useful accounts of the history and epidemiology of poliomyelitis at various periods, particularly in England and Wales, have been given by Macewen (1912), Low (1915-16), McNalty (1936), Rhodes (1947), Gale (1949, 1951a, b) and Richmond (1951).

In 1947 a new phase opened in the epidemic history of poliomyelitis in this country. More than five times as many cases were reported in that year as in either of the two previously worst years 1926 and 1938. Two years later, in 1949, poliomyelitis again attained this new high prevalence, and once more for the third time in 1950 (Figure 1). Even in 1948 and 1951, non-epidemic years by recent standards, more cases were notified than in any year prior to 1947 ; though some of the prevalence of these two years represented merely the tail of the previous year's epidemics, augmented possibly by a wider realisation among physicians of the likelihood of the disease occurring within their practice and of its main characteristics, so that fewer cases than formerly would escape notification. In addition to the official reports for the years 1947 to 1950 of the Chief Medical Officer of the Ministry of Health and of the Registrar General, the epidemic of 1947 has been described from the national viewpoint by Gale (1948) and by Gunn (1948), and a general account covering 1947 to 1950 has been given by Martin (1951).

Three times therefore in the past few years a disease which, however disastrous to its occasional victims and grievous to their relatives, had been of small epidemiological consequence in this country has assumed serious epidemic proportions and become a cause of much Public Health concern. This concern has been all the more increased by the realisation that in

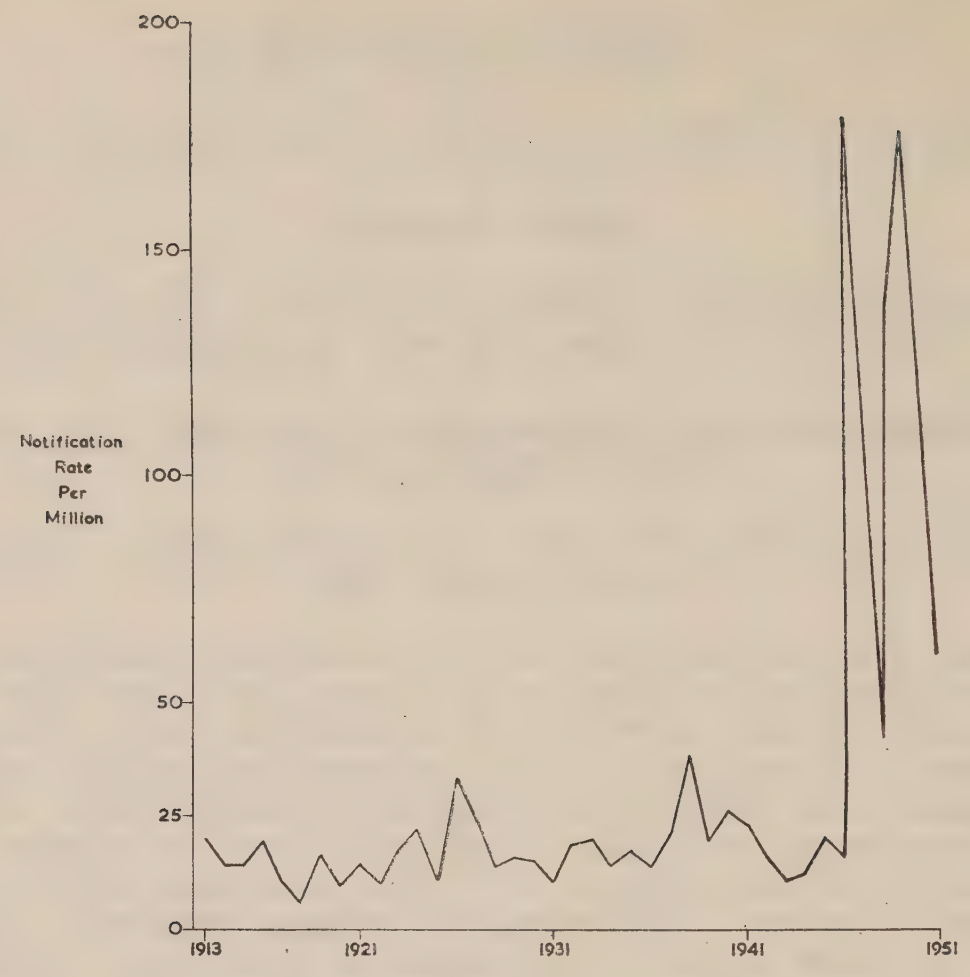


FIG. 1

Poliomyelitis. Notification rates per million persons. England and Wales, 1913 to 1951 (corrected notifications, 1944 to 1951).

countries, notably the United States of America, that have already had many years' experience of epidemic poliomyelitis on a considerable scale, a vast amount of assiduous research and endeavour has not yet led to effective measures of prevention or of cure. "In recent years a knowledge of the virus and of the pathology of poliomyelitis has been considerably increased by work done abroad, but we cannot yet state with any certainty the mode in which infection is conveyed to the sufferer, and no specific remedy for the disease is known." (Local Government Board, 1911-12.) Unfortunately these words are nearly as true to-day as they were forty years ago, despite accumulating knowledge about the virus of poliomyelitis and its ways.

Why poliomyelitis should have suddenly assumed a new epidemic character in this country in 1947 is one of the all too numerous epidemiological riddles to which no ready answer can be found. Even the traditional expedient of placing the source of an epidemic in a neighbouring country (e.g. French pox, Spanish 'flu') has found no proponents for the poliomyelitis of 1947-50; though the possibility cannot be dismissed that a new strain (new that is to this country) of poliomyelitis virus gained entrance during the war years when large numbers of foreign troops were temporarily stationed within the country, but had to wait until 1947 to find conditions suitable for the production of a widespread epidemic. Another traditional expedient is to blame the weather, and it has been frequently suggested that the warm summers of 1947 and 1949 may have had something to do with the epidemics of these years; it is true that summer temperatures of 1947 and of 1949 were among the highest recorded in this century, exceeded only in 1911 and 1933, but this association may be no more than coincidental. Nevertheless in view of the regular seasonal variations in the incidence of poliomyelitis within each year, some relationship between meteorological conditions and poliomyelitis epidemicity no doubts exists.

Definitions

Throughout this paper "notifications" mean *corrected* notifications, i.e., notifications that have been corrected for subsequent amendments to diagnosis by the notifying medical practitioner or by the medical superintendent of the infectious diseases hospital. Unless otherwise indicated notifications prior to 1950 include cases notified either as poliomyelitis or as polioencephalitis; similarly, deaths from poliomyelitis include deaths from polioencephalitis. From the beginning of 1950 when new regulations were introduced whereby polioencephalitis ceased to be a separately notifiable disease, notifications include cases notified either as paralytic or as non-paralytic poliomyelitis.

In the various tables notifications and deaths of non-civilians stationed in England and Wales have been included from 1950 onwards but have been excluded in previous years.

Fatality rates are numbers of deaths registered in a given period per hundred corrected notifications during the same period. They are ratios rather than rates.

The *median age* of notified cases is the calculated age of the middle case if all the cases were arranged in ranking order from youngest to oldest. This measure has some advantage over the more familiar arithmetic mean or average in that it is easier to calculate and is less affected by a few cases at extreme ages.

The geographical regions referred to are the Standard Regions. The constitution of these regions will be found in the explanatory notes in any recent volume of the Registrar General's Statistical Review.

Notification, death and fatality rates, 1947-1950

TABLE 1

Poliomyelitis: Mean annual notification, death and fatality rates by sex and age. England and Wales, 1947–50

	All Ages	0–	1–	3–	5–	10–	15–	25 and over
Males								
Notification rate per 100,000 ...	16	32	66	65	49	31	18	4
Death rate per million	16	40	34	31	30	23	28	9
Fatality rate (deaths per cent. of notifications)	10·4	12·3	5·2	4·8	6·1	7·5	15·2	25·5
Females								
Notification rate per 100,000 ...	12	28	57	53	35	23	14	3
Death rate per million	11	36	27	22	22	16	20	6
Fatality rate (deaths per cent. of notifications)	9·7	12·7	4·8	4·2	6·1	6·7	14·3	19·3

The mean annual notification rate of males at all ages was 157 per million and of females 116 per million. Males therefore ran a one-third greater risk of contracting recognisable poliomyelitis than females. The distribution of case rates at different ages was the same in each sex, highest at ages 1 and 2 followed closely by ages 3 and 4 and then ages 5 to 9. The rate under one year just exceeded that at 10–14 and next came ages 15–24 and 25 and over (Figure 2). At each age the male rate was higher than the female and the sex ratio rose to a maximum at ages 5–9 declining a little at higher ages, as is shown by the percentage ratio of male to female rates at each age:—

All ages	0–	1–	3–	5–	10–	15–	25+
133	114	116	123	140	135	129	133

The mean annual death rate of males at all ages in 1947–50, 16·3 per million, was 45 per cent. higher than the female, 11·2 per million, but in

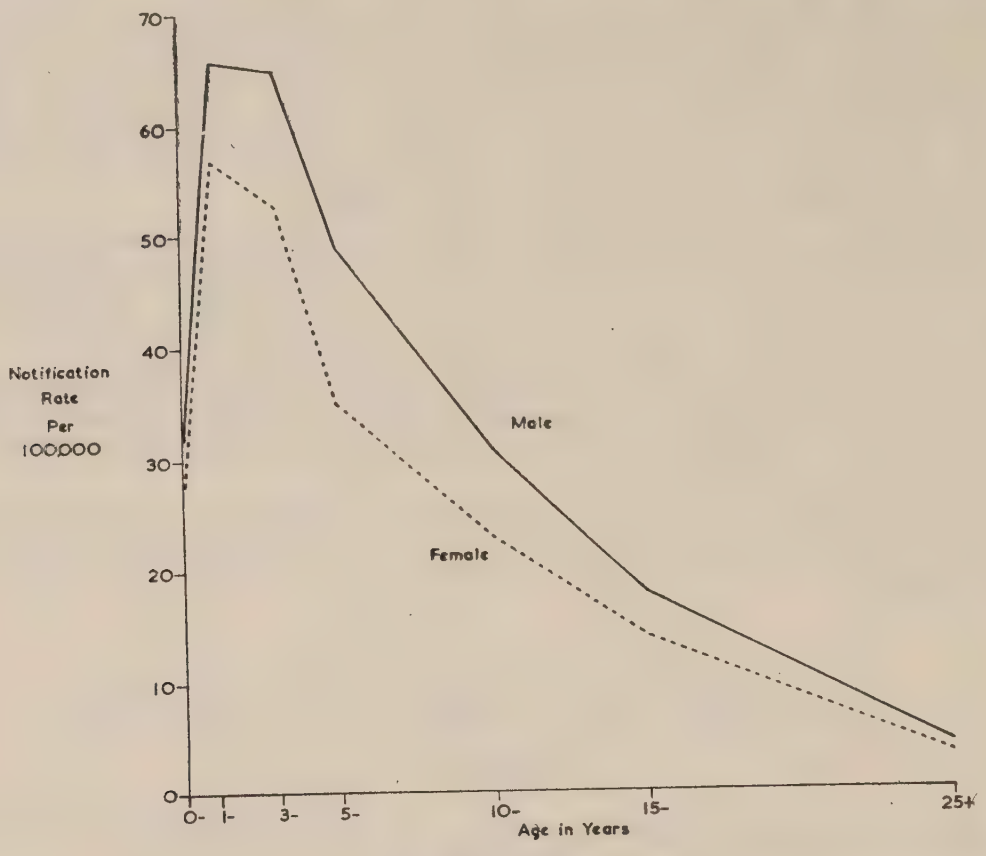


FIG. 2

Poliomyelitis. Mean annual notification rates, by sex and age. England and Wales, 1947–50.

contrast with notifications, the death rate was highest in infancy and diminished with increasing age, the only irregularity in this progression being at ages 10–14 which had lower mortality in each sex than ages 15–24.

The sex ratios of mortality (male rates per cent. of female) were:—

All ages	0–	1–	3–	5–	10–	15–	25+
145	111	126	141	136	144	140	150

The fatality rate was about 5 per cent. in each sex at ages 1–4, slightly higher at 5–14, and higher still in infancy and at ages 15 and over. There was one death for every four cases notified among males aged 25 and over. At most ages fatality was slightly higher among males than among females, the rates at all ages being 10·4 for males and 9·7 for females.

Even during this period of high prevalence poliomyelitis was responsible for fewer deaths of children than whooping cough or measles. Annual death rates from these three causes per million children aged under 15 are compared in the next table:—

	1947	1948	1949	1950
Poliomyelitis	33	8	29	33
Whooping cough... ...	99	80	55	41
Measles	69	33	30	22

Urban—Rural Differences

TABLE 2

Poliomyelitis. Notification, death and fatality rates by sex and age. Density Aggregates, 1947 to 1950.

		Notifications per 100,000			Deaths per million			Deaths per cent. of notifications		
		0—	5—	15 and over	0—	5—	15 and over	0—	5—	15 and over
Greater London										
Males	1947	74	80	9	26	55	13	3·5	6·8	13·7
	1948	19	17	2	3	14	8	1·5	8·1	31·5
	1949	100	54	6	42	39	11	4·2	7·3	17·5
	1950	53	47	6	14	23	10	2·7	5·0	16·6
Females ...	1947	53	54	9	27	25	8	5·2	4·6	9·4
	1948	14	13	3	6	2	6	4·3	1·5	22·6
	1949	81	44	5	45	24	11	5·5	5·6	22·2
	1950	49	33	6	15	12	7	3·0	3·6	10·9
County Boroughs										
Males	1947	70	41	7	40	19	11	5·7	4·7	16·0
	1948	19	10	2	7	8	4	3·6	7·5	26·6
	1949	59	32	4	49	18	10	8·3	5·6	22·3
	1950	99	56	7	66	24	13	6·6	4·3	20·3
Females ...	1947	60	32	6	27	14	9	4·5	4·4	14·5
	1948	17	8	1	5	6	2	3·2	7·1	20·7
	1949	48	23	4	28	18	9	5·9	7·8	22·0
	1950	90	40	6	49	34	7	5·4	8·4	12·8
Other Urban Districts										
Males	1947	77	50	7	55	45	15	7·2	9·1	20·0
	1948	17	11	2	3	14	6	1·9	12·1	34·5
	1949	58	38	6	32	35	14	5·5	9·3	24·1
	1950	67	46	8	48	30	15	7·2	6·4	20·3
Females ...	1947	68	40	6	38	28	10	5·6	7·1	15·7
	1948	14	7	2	11	4	4	7·8	6·7	22·7
	1949	46	26	4	35	24	9	7·7	9·1	20·1
	1950	61	32	6	35	17	13	5·7	5·2	19·9
Rural Districts										
Males	1947	72	71	11	44	30	24	6·1	4·2	21·1
	1948	18	12	3	6	12	8	3·2	10·0	27·9
	1949	48	42	7	20	36	16	4·2	8·6	22·7
	1950	77	56	10	71	32	17	9·2	5·7	17·3
Females ...	1947	55	45	8	28	30	10	5·1	6·8	12·4
	1948	12	10	2	9	11	5	7·9	11·3	24·6
	1949	44	34	6	24	15	12	5·5	4·2	20·6
	1950	63	49	9	39	34	16	6·2	6·9	17·1

Mean annual notification rates per 100,000 in the main density aggregates in 1947–50 (both sexes) were as follows:—

	Age		
	0—	5—	15+
Greater London	56	43	6
County Boroughs	58	30	4
Other Urban Districts ...	51	31	5
Rural Districts	49	40	7

The combined County Boroughs had the highest rate at ages under 5 (58 per 100,000) and the lowest rate at ages 15 and over (4 per 100,000). The Rural Districts had the lowest rate under 5 (49) and the highest rate at 15 and over (7). There was thus a reversal of the relative position of these two classes of area at these ages (Figure 3), and it is unlikely that this could have arisen from differing degrees of completeness of notification in the different areas.

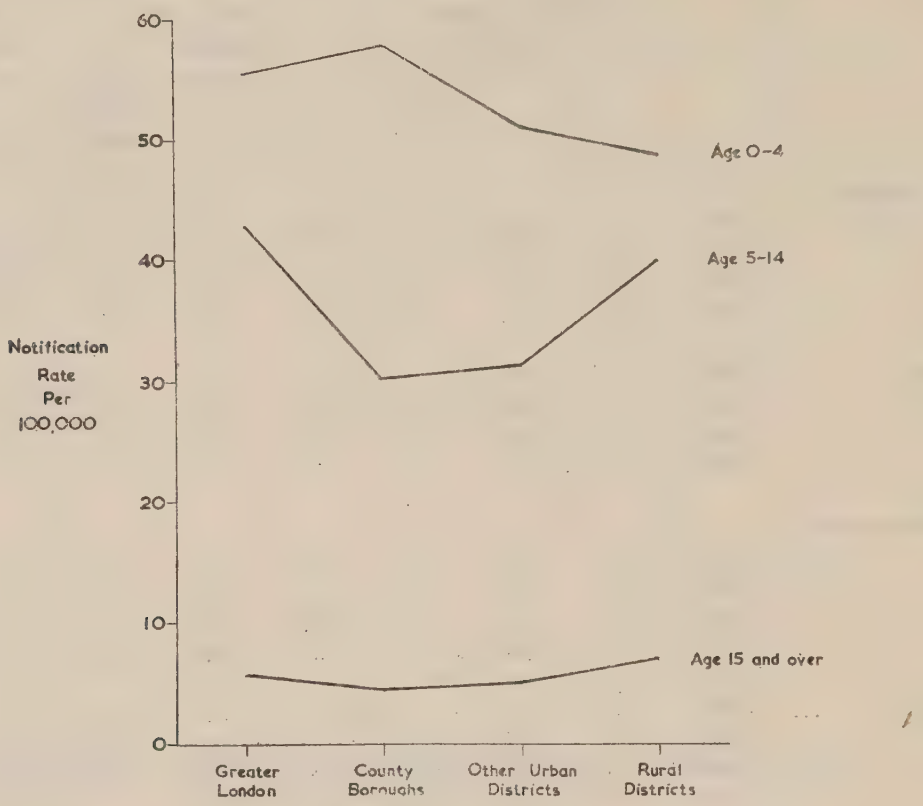


FIG. 3

Poliomyelitis. Mean annual notification rates, by age. Density aggregates of England and Wales, 1947–50.

In each sex the notification rate in Greater London under 5 was exceptionally high in 1949 compared with older ages.

Mean annual death rates per million in 1947–50 in the four classes of area were as follows:—

	Age		
	0—	5—	15+
Greater London	22	24	9
County Boroughs	34	18	8
Other Urban Districts ...	32	25	10
Rural Districts	30	25	14

The County Boroughs had the highest rate at ages under 5 and the lowest rate at higher ages. The death rate in Greater London was lower under 5 than at 5–9 in contradistinction to the other areas where the death rate was highest in the youngest age group.

Fatality rates (deaths per cent. of notifications) at these three ages during 1947–50 were:—

	Age		
	0—	5—	15+
Greater London	4·0	5·7	15·6
County Boroughs	5·9	5·8	18·0
Other Urban Districts ...	6·3	7·9	20·6
Rural Districts	6·2	6·3	19·0

At each age fatality was lowest in Greater London and highest in the Urban Districts. In County Boroughs the rate was higher at ages 0–4 than at 5–14.

TABLE 3A

Poliomyelitis. Notifications, Notification rates and rates per cent. of England and Wales. Standard Regions and Density Aggregates, 1947 to 1950.

	Numbers of Notifications				Notification rates per 100,000				Notification rates per cent. of England and Wales			
	1947	1948	1949	1950	1947	1948	1949	1950	1947	1948	1949	1950
England and Wales	7,646	1,829	5,918	7,750	18	4	14	18	100	100	100	100
Northern	688	72	206	578	23	2	7	18	128	53	50	100
East and West Ridings	830	137	734	574	21	3	18	14	117	79	129	78
North Western	998	185	563	628	16	3	9	10	89	67	64	56
North Midland	573	166	497	650	18	5	15	19	100	118	107	106
Midland	709	151	286	1,482	17	4	7	33	94	81	50	183
Eastern	492	146	503	439	17	5	17	14	94	114	121	78
London and South Eastern	2,170	534	1,860	1,518	21	5	17	14	117	116	121	78
Southern	462	143	485	535	19	6	19	20	106	133	136	111
South Western	354	172	587	938	12	6	20	31	67	137	143	172
Wales	370	123	197	408	15	5	8	16	83	112	57	89
Greater London	1,674	430	1,482	1,176	21	5	18	14	117	121	129	78
County Boroughs	1,992	506	1,531	2,595	16	4	12	20	89	91	86	111
Other Urban Districts	2,334	519	1,753	2,273	18	4	13	16	100	88	93	89
Rural Districts	1,646	374	1,152	1,706	21	5	14	21	117	109	100	117

TABLE 3B

Poliomyelitis. Deaths, Death rates, and rates per cent. of England and Wales. Standard Regions and Density Aggregates, 1947 to 1950.

	Numbers of Deaths				Death rates per million				Death rates per cent. of England and Wales			
	1947	1948	1949	1950	1947	1948	1949	1950	1947	1948	1949	1950
England and Wales	688	239	651	734	16	6	15	17	100	100	100	100
Northern	74	13	21	45	25	4	7	14	156	75	47	82
East and West Ridings	60	19	103	48	15	5	25	12	94	84	167	71
North Western	107	28	80	66	17	4	12	10	106	79	80	59
North Midland	57	18	53	68	18	6	16	20	113	98	107	118
Midland	54	20	29	159	13	5	7	36	81	82	47	212
Eastern	59	21	51	41	21	7	17	13	131	127	113	76
London and South Eastern	186	76	191	115	18	7	18	10	113	127	120	59
Southern	44	13	46	54	18	5	18	20	113	93	120	118
South Western	15	19	56	104	5	7	19	35	31	116	127	206
Wales	32	12	21	34	13	5	8	13	81	84	53	76
Greater London	124	55	134	86	15	7	16	10	94	118	107	59
County Boroughs	162	52	170	222	13	4	13	17	81	72	87	100
Other Urban Districts	244	74	215	244	18	5	16	18	113	97	107	106
Rural Districts	158	58	132	182	20	7	17	22	125	130	113	129

Geographical Differences in Rates at all ages

In 1947 notifications and deaths were highest in the Northern region, the notification rate being 28 per cent. and the death rate 56 per cent. above the rate for England and Wales (Tables 3A and 3B). In the South Western region the notification rate was 33 per cent. below and the death rate 69 per cent. below the national average. In the density analysis, Greater London and the aggregated Rural Districts each had notification rates 17 per cent. above the national average, but whereas the death rate in Greater London was 6 per cent. below average that of the Rural Districts was 25 per cent. above average for the country as a whole.

In 1948, in which prevalence was generally much lower than in the preceding and the two following years, the Northern region had the lowest notification and death rates. Rates in Greater London and the Rural Districts were higher than in the County Boroughs and the other Urban Districs.

In 1949 the Northern and the Midland regions had lowest notification and death rates. The region with the highest notification rate was the South Western, 43 per cent. above the national average, whereas the highest death rate, 67 per cent. above average, was recorded in the East and West Ridings of Yorkshire. Greater London again gave a notification rate well above the average, and again the highest death rate of the four density groups was in the Rural Districts.

In 1950 notifications in the South Western region were once more well above the national average but a still higher rate was recorded in the Midland region, 83 per cent. above average. In both of these regions the death rates were more than double that of the country as a whole. Once more the notification and death rates of the Rural Districts were much in excess of those in other types of area. It will be noted that the Northern, South Western, and Midland regions each had the highest notification rate in one of the three epidemic years and the lowest rate in another of these years.

Percentage Age Distribution and Median Age of Notified Cases England and Wales

Table 4A gives numbers and percentage age distribution of notified cases each year from 1944 to 1950. On average over the whole period 20 per cent. of cases in each sex were at ages under 3 and 14 per cent. at ages 3 and 4. Thus a third of the total cases were in children under 5. Approximately a

FIG. 4

Poliomyelitis. Median age of notified cases, by sex. England and Wales, 1944 to 1950.

further third were at ages 5 to 14 and the remaining third at ages 15 and over, the proportion at adult ages being higher among women than men.

The median age of notified cases each year from 1944 to 1950 and in each quarter of the year is shown in Table 4B. Over the whole period the median age of male cases was 8·46 years and of female cases 9·15 years. It should be noted, however, that the median age of the male population during the period at ages under 25 (civilians only, 1944–49) was considerably lower than that of females, 10·64 years for males compared with 12·24 for females. The annual median age of male cases was highest in 1945, 9·32 years, and lowest in 1949, 7·90 years. For females the highest annual median age was in 1948, 10·17 years, and, as for males, lowest in 1949, 8·32 years (Figure 4).

The quarterly variations of age presented a peculiar feature, namely a tendency for the median age to be highest in the first quarter and lowest in the second quarter of the year ; i.e., the two quarters in which prevalence was invariably least. The first quarter gave the highest median age on four occasions for each sex, and the second quarter gave the lowest median age

TABLE 4A

Poliomyelitis. Number and Percentage Age Distribution of notified cases, by sex. England and Wales, 1944 *to* 1950.

		All Ages	0–	1–	3–	5–	10–	15–	25+	Unknown
Males										
1944	... No.	257	5	31	47	66	47	31	25	5
	Percentage	100	2	12	18	26	18	12	10	2
1945	... No.	432	19	77	37	92	68	67	65	7
	Percentage	100	4	18	9	20	16	16	15	2
1946	... No.	333	16	44	39	78	52	62	40	2
	Percentage	100	5	13	12	22	16	19	12	1
1947	... No.	4,227	163	602	576	964	641	611	636	34
	Percentage	100	4	14	14	23	15	14	15	1
1948	... No.	1,020	53	157	130	202	152	161	159	6
	Percentage	100	5	15	13	19	15	16	16	1
1949	... No.	3,289	131	600	481	734	431	450	447	15
	Percentage	100	4	18	15	22	13	14	14	0
1950	... No.	4,219	163	656	630	1,027	508	560	656	19
	Percentage	100	4	16	15	24	12	13	16	0
1944–50	... No.	13,777	550	2,067	1,940	3,163	1,899	1,942	2,028	88
	Percentage	100	4	16	14	22	14	14	15	1
Females										
1944	... No.	238	4	39	29	54	36	35	36	5
	Percentage	100	2	16	12	23	15	15	15	2
1945	... No.	389	18	52	45	91	56	71	51	5
	Percentage	100	5	13	12	24	14	18	13	1
1946	... No.	322	10	40	38	76	44	48	61	5
	Percentage	100	3	12	12	23	14	15	19	2
1947	... No.	3,419	139	474	438	642	473	607	622	24
	Percentage	100	4	14	13	18	14	18	18	1
1948	... No.	809	32	132	90	144	103	128	174	6
	Percentage	100	4	16	11	18	13	16	21	1
1949	... No.	2,629	125	451	371	547	302	381	443	9
	Percentage	100	5	17	14	22	11	14	17	0
1950	... No.	3,531	129	591	519	701	400	541	638	12
	Percentage	100	4	17	15	20	11	15	18	0
1944–50	... No.	11,337	457	1,779	1,530	2,255	1,414	1,811	2,025	66
	Percentage	100	4	16	14	19	12	16	18	1

TABLE 4B

Poliomyelitis. Quarterly Numbers and Median Age of Notified Cases, by sex. Median Age of Population aged under 25. England and Wales, 1944 to 1950.

	1st Quarter	2nd Quarter	3rd Quarter	4th Quarter	Year*	Median Age of population under 25
Males						
1944 No.	37	47	100	72	257	
Median Age	8·27	6·88	8·46	8·83	8·26	*10·20*
1945 No.	30	45	177	178	432	
Median Age	8·61	9·32	7·80	11·76	9·32	*9·97*
1946 No.	39	45	132	117	333	
Median Age	11·50	7·69	10·54	8·17	9·26	*10·38*
1947 No.	48	148	2,947	1,108	4,227	
Median Age	12·50	7·88	8·91	9·00	8·92	*10·58*
1948 No.	176	103	359	387	1,020	
Median Age	11·36	7·95	8·21	8·70	9·13	*11·04*
1949 No.	91	123	1,455	1,619	3,289	
Median Age	11·03	7·73	7·91	7·70	7·90	*10·94*
1950 No.	212	328	2,576	1,104	4,219	
Median Age	7·96	6·72	8·45	8·10	8·17	*11·38*
Females						
1944 No.	30	42	96	70	238	
Median Age	10·00	5·31	11·41	8·16	9·12	*12·77*
1945 No.	26	34	147	182	389	
Median Age	8·93	8·08	9·17	9·74	9·23	*12·53*
1946 No.	53	38	125	105	322	
Median Age	10·42	11·07	10·50	8·00	9·64	*12·58*
1947 No.	52	126	2,350	892	3,419	
Median Age	13·57	7·27	9·91	10·91	10·05	*12·22*
1948 No.	148	79	317	271	809	
Median Age	14·90	12·68	8·28	9·74	10·17	*12·02*
1949 No.	80	103	1,190	1,259	2,629	
Median Age	10·00	9·02	8·18	8·33	8·32	*11·86*
1950 No.	196	284	2,055	998	3,531	
Median Age	11·58	6·81	8·71	8·88	8·71	*11·75*

* Owing to occasional amendments to annual totals they do not necessarily correspond exactly to the sum of the four quarters.

five times for males and four times for females. This can be further illustrated by averaging the quarterly median ages shown in Table 4B, thus:—

	Quarter			
	1st	2nd	3rd	4th
Median age of male cases	10·18	7·74	8·61	8·89
Median age of female cases ...	11·34	8·61	9·45	9·11

If, however, the second quarter is regarded as the opening phase of each year's epidemicity, and the first quarter as the closing phase of that of the previous year, the figures indicate a progressive rise in the median age throughout the course of the annual epidemic cycle. Sutherland (1951) has drawn attention to a similar sort of thing in Scotland.

Age-shift

It has been frequently pointed out (e.g., Benjamin and Gale, 1949) that there has been a pronounced shift in the age distribution of notified poliomyelitis in recent years compared with twenty or thirty years ago, an age-shift much greater than could have been brought about by changes in the age structure of the population. To illustrate this phenomenon the following table compares the percentage age distribution and median age of notifications in 1912–19 and 1944–50:—

		Age			All Ages	Median Age
		0–4	5–14	15+		
1912–19	M	64	28	8	100	3·93
	F	66	28	6	100	3·90
1944–50	M	34	37	29	100	8·46
	F	33	33	34	100	9·15

In contrast with the median age of notification of 8·46 years for males in 1944–50 the median age of male notification in 1912–19 was 3·93 years. For females the median age of notification was 9·15 years in 1944–50 but only 3·90 in 1912–19. As with several other infectious diseases where a similar kind of age-shift has been observed, e.g., diphtheria, measles, scarlet fever, a possible explanation is that improvements in environmental hygiene and in standards of living, smaller family size and reduction in home crowding have tended to diminish the opportunities for infection among very young children ; but detailed understanding of the mechanisms involved is lacking. Alternatively, new strains of virus may be the explanation (Weinstein *et al.*, 1952).

Geographical Variations in Age Distribution

(a) *Standard Regions* (Tables 5A and 5B)

For the combined five-year period 1946–50 the median age of notified cases of each sex was highest in the Southern and Eastern regions followed for males by the North Midland region and for females by London and the South-East. The regions with the lowest median age of notifications for each sex were the Northern, the North Western and Wales. These regional variations are quite unrelated to the median age of the regional populations under 25 or to latitude, but follow very closely, though inversely, the regional pattern of infant mortality rates, these being highest in the north of England and in Wales and lowest in the Southern and Eastern parts of the country (Figure 5). This finding may be construed as perhaps giving some support to a hypothesis that higher age attack is associated with better social circumstances ; but too much should not be made of this observation at present and the matter requires further study.

There is also some suggestion of correlation between the median age of notified cases in the various regions and the average notification rates for the period 1946–50. The three regions Northern, North Western, and Wales,

TABLE 5A

Poliomyelitis. Median Age of Notified Cases, and Percentage Age Distribution. Standard Regions, Males, 1946 *to* 1950.

	Median Age of Notified Cases						Median Age of Population under 25	Mean Annual Rate per 100,000	Total Number Notified	Percentage Age Distribution, 1946–50								
	1946	1947	1948	1949	1950	1946–50	1947–50	1946–50	1946–50	All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown
Northern	5·00	7·21	4·33	8·47	5·96	6·70	*11·52*	11	845	100	5	23	16	19	14	13	9	1
East and West Ridings ...	7·50	9·03	8·33	7·81	7·66	8·15	*11·01*	13	1,257	100	4	18	14	22	13	14	15	0
North Western	9·62	6·03	7·07	6·75	5·40	6·29	*10·96*	9	1,313	100	5	22	17	22	12	11	11	0
North Midland	5·83	11·30	9·87	9·73	8·84	9·67	*11·18*	13	1,063	100	3	13	13	21	15	16	18	1
Midland	12·50	9·00	9·13	7·83	6·83	7·65	*10·93*	14	1,493	100	4	17	15	26	12	12	14	0
Eastern	9·29	9·67	11·25	9·30	10·69	9·85	*11·19*	13	939	100	3	9	13	26	16	16	17	0
London and South Eastern	10·50	9·35	9·85	7·33	9·44	8·87	*10·96*	14	3,435	100	4	14	14	24	14	14	16	0
Southern	10·83	11·38	9·52	9·41	12·34	10·90	*11·39*	16	926	100	3	10	12	23	17	17	18	0
South Western	9·44	9·92	10·00	7·69	8·18	8·39	*11·29*	17	1,185	100	5	15	14	23	13	17	12	1
Wales	4·20	7·84	7·50	4·80	7·33	6·84	*11·48*	10	632	100	5	22	14	18	11	12	14	4

TABLE 5B

Poliomyelitis. Median Age of Notified Cases, and Percentage Age Distribution. Standard Regions, Females, 1946–50

	Median Age of Notified Cases						Median Age of Population under 25	Mean Annual Rate per 100,000	Total Number Notified	Percentage Age Distribution, 1946–50								
	1946	1947	1948	1949	1950	1946–50	1947–50	1946–50	1946–50	All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown
Northern	5·00	5·87	10·00	9·06	5·70	6·26	*12·11*	9	747	100	6	24	16	17	11	11	14	1
East and West Ridings ...	12·50	8·87	4·83	7·98	7·50	8·10	*11·84*	10	1,042	100	4	18	16	18	12	15	17	0
North Western	7·50	7·47	6·88	7·21	6·18	7·03	*11·97*	7	1,141	100	6	22	15	17	11	14	15	0
North Midland	14·17	11·03	8·98	8·00	8·77	9·25	*11·82*	10	864	100	3	16	13	21	13	17	17	0
Midland	7·50	9·77	6·92	7·07	5·17	7·82	*11·72*	11	1,199	100	5	16	17	21	11	13	17	0
Eastern	10·00	11·37	9·26	11·76	13·07	11·85	*11·89*	9	691	100	2	11	9	23	14	18	23	0
London and South Eastern	12·03	12·41	13·89	7·76	11·03	10·56	*12·28*	10	2,842	100	3	12	13	20	14	18	20	0
Southern	8·75	12·59	13·18	10·98	13·31	12·31	*11·91*	11	745	100	3	11	10	19	14	19	23	1
South Western	9·81	13·91	9·62	9·65	8·19	9·38	*12·01*	12	937	100	4	15	11	23	11	15	20	1
Wales	6·67	9·05	8·13	4·20	7·99	7·47	*12·03*	8	502	100	6	18	16	18	11	15	14	2

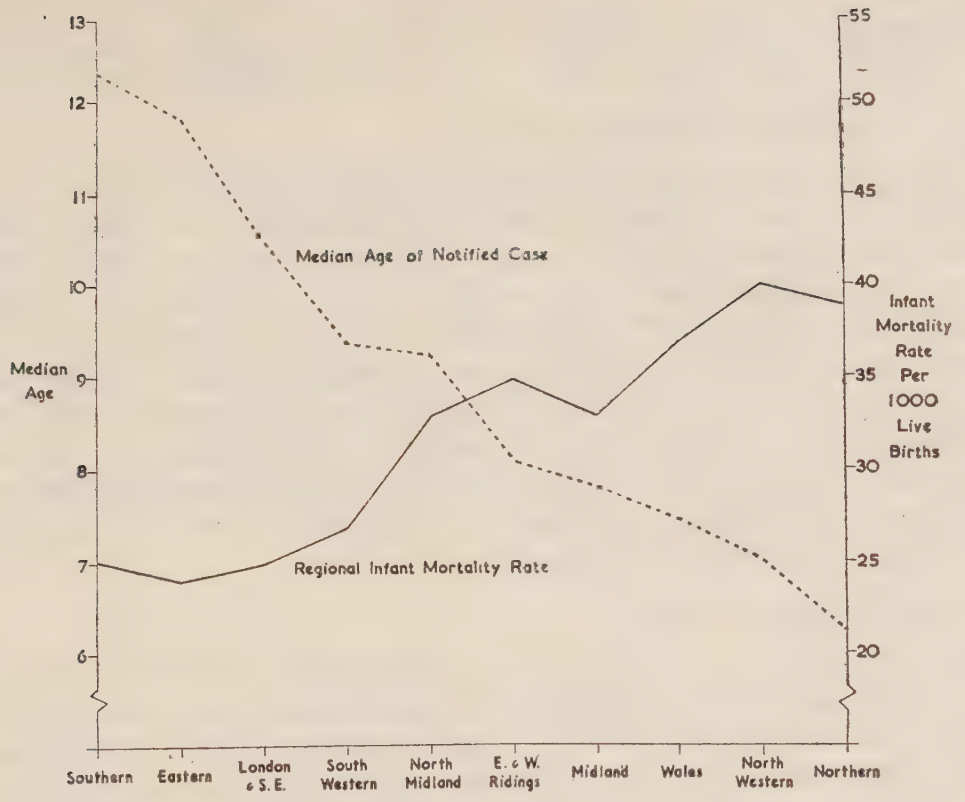


FIG. 5

Poliomyelitis. Median age of notified cases. Infant Mortality Rates. Females. Standard Regions of England and Wales, 1946–50.

which were the regions with lowest median age of attack, were also the three regions with lowest average incidence of notified poliomyelitis during the period. Nevertheless the median age of notifications in individual regions did not fluctuate much from year to year with variations in incidence. There was in fact a general tendency for each region to show a median age of attack that was regularly either above or below average for the country as a whole. This is illustrated in Table 6 which sets out the ranking order of the regions from year to year according to median age of notifications. There is a fair degree of concordance between the ranking orders for the various years and it is unlikely to have arisen by chance. (Coefficient of concordance in each sex, W = 0·58 ; P less than 0·01.)

TABLE 6

Poliomyelitis. Ranking Order of Standard Regions by Median Age of Notified Cases. 1946 *to* 1950.

	Male					Female				
	1946	1947	1948	1949	1950	1946	1947	1948	1949	1950
Northern	9	9	10	4	9	10	10	3	4	9
E. & W. Ridings...	7	6	7	6	6	2	8	10	6	7
North Western ...	4	10	9	9	10	7½	9	9	8	8
North Midland ...	8	2	3	1	4	1	5	6	5	4
Midland	1	7	6	5	8	7½	6	8	9	10
Eastern	6	4	1	3	2	4	4	5	1	2
London and South-East	3	5	4	8	3	3	3	1	7	3
Southern	2	1	5	2	1	6	2	2	2	1
South Western ...	5	3	2	7	5	5	1	4	3	5
Wales	10	8	8	10	7	9	7	7	10	6

It has already been noted that for the country as a whole the median age of notification for each sex was lowest in 1949. A region that had a notably low age of notification in that year was London and the South-East.

(b) *Density Aggregates* (Tables 7A and 7B)

In general the median age of notified cases of each sex was lowest in the County Boroughs and highest in the Rural Districts. In 1949, however, the notifications in Greater London gave the lowest median age. The exceptionally low age distribution of cases in London in 1949 has already been commented on by Benjamin and Breen (1950). Although for males the general age relationship of notifications resembled that of the population under 25 (County Boroughs lowest, Rural Districts highest) this correlation was entirely absent with regard to females, the median age of the female population being lowest in the Rural Districts and highest in Greater London and the County Boroughs. In each of the four areas the median age for males was lower than that for females.

(c) *Seven largest cities* (Table 8)

The median age of notifications of males in 1946–50 was highest in Sheffield and Leeds, while for females it was highest in Leeds and London. Notifications in Liverpool gave the lowest median age for each sex. The variations in median age of notifications among the seven cities were quite unrelated to the age composition of their populations or to their average annual notification rates. Sheffield was the only city in which the median age of male cases was higher than of female cases.

Poliomyelitis and Polioencephalitis
(*Table* 9)

As already explained, from 1919 to 1949 poliomyelitis was statutorily notifiable under the designation either of poliomyelitis or of polioencephalitis.

During 1946–49 8·2 per cent. of male cases and 7·2 per cent. of female cases were notified as polioencephalitis, the percentages varying with age in the following way:—

	0–	1–	3–	5–	10–	15–	25+
Male ...	8·6	6·6	7·2	8·5	9·6	7·1	10·1
Female	7·9	4·4	6·8	6·1	6·4	7·4	11·2

The age distribution and median age of notifications of poliomyelitis and of polioencephalitis are compared in Table 9. For both types of disease the median age of female cases was higher than of male cases. In each sex, but particularly among females, the median age of notifications of polioencephalitis was higher than of poliomyelitis (Figure 6). Forty-five per cent. of female cases notified as polioencephalitis were at ages 15 and over, compared with 31 per cent. of male polioencephalitis, 33 per cent. of female poliomyelitis, and 29 per cent. of male poliomyelitis.

The sex ratios (male notification rate per cent. of female) at each age were:—

	0–	1–	3–	5–	10–	15–	25+	All ages
Poliomyelitis	111	119	123	132	129	141	110	137
Polioencephalitis ...	125	182	132	193	200	137	104	159

TABLE 7A

Poliomyelitis. Median Age of Notified Cases, and Percentage Age Distribution. Density Aggregates, Males, 1946 to 1950.

	Median Age of Notified Cases						Median Age of Population under 25	Mean Annual Rate per 100,000	Total Number Notified	Percentage Age Distribution, 1946–50								
	1946	1947	1948	1949	1950	1946–50	1947–50	1946–50	1946–50	All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown
Greater London	9·55	9·17	9·65	6·54	8·71	8·38	*10·99*	14	2,676	100	4	15	14	26	13	13	15	0
County Boroughs	8·88	8·05	8·11	7·08	6·72	7·30	*10·96*	12	3,690	100	5	20	15	21	13	13	13	0
Other Urban Districts ...	8·37	8·20	8·25	8·35	8·57	8·37	*11·03*	12	3,906	100	4	16	14	23	13	14	15	1
Rural Districts	10·96	10·51	11·59	9·78	9·52	10·05	*11·62*	15	2,816	100	3	11	13	22	15	17	17	2

TABLE 7B

Poliomyelitis. Median Age of Notified Cases, and Percentage Age Distribution. Density Aggregates, Females, 1946 to 1950

	Median Age of Notified Cases						Median Age of Population under 25	Mean Annual Rate per 100,000	Total Number Notified	Percentage Age Distribution, 1946–50								
	1946	1947	1948	1949	1950	1946–50	1947–50	1946–50	1946–50	All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown
Greater London	11·15	12·25	13·47	6·95	10·33	9·89	*12·43*	10	2,238	100	4	13	13	21	13	18	18	0
County Boroughs	7·13	8·90	7·07	7·92	6·53	7·55	*12·04*	9	3,109	100	5	19	16	19	11	14	16	0
Other Urban Districts ...	12·03	9·12	9·23	8·65	9·35	9·16	*11·87*	9	3,175	100	4	16	13	20	12	15	19	1
Rural Districts	8·97	11·30	12·50	10·10	10·36	10·70	*11·76*	11	2,188	100	3	12	11	21	14	18	20	1

TABLE 8

Poliomyelitis. Median Age of Notified Cases, and Percentage Age Distribution. Seven largest Towns, 1946–50.

	Median Age of Notified Cases	Median Age of Population under 25	Mean Annual Rate per 100,000	Total Number Notified	Percentage Age Distribution, 1946–50							
	1946–50	1947	1946–50	1946–50	All Ages	0–	1–	3–	5–	10–	15–	25 and over
MALES												
London Administrative County	6·92	10·94	14	1,077	100	6	21	14	24	11	11	13
Birmingham	7·56	10·13	17	446	100	4	17	15	27	15	10	12
Liverpool	4·20	10·70	8	136	100	7	32	18	14	10	9	10
Manchester	5·43	10·76	10	160	100	8	25	15	22	9	11	10
Sheffield...	8·68	10·14	14	178	100	3	17	14	22	15	13	16
Leeds	8·46	10·61	13	148	100	4	22	11	18	14	14	17
Bristol	6·08	10·71	27	270	100	6	22	17	22	9	13	11
FEMALES												
London Administrative County	8·71	13·28	10	914	100	5	18	13	19	11	17	17
Birmingham	7·58	11·58	13	383	100	4	14	18	27	9	11	17
Liverpool	4·72	12·34	7	139	100	9	29	13	15	12	8	14
Manchester	7·26	12·56	7	127	100	4	21	17	17	10	17	14
Sheffield...	6·40	11·47	12	161	100	6	20	19	20	9	14	12
Leeds	9·67	12·34	9	114	100	4	20	14	13	10	18	21
Bristol	6·63	12·22	17	198	100	5	23	15	22	13	9	13

TABLE 9

Notifications of Poliomyelitis and of Polioencephalitis. Numbers, Percentage Age Distribution, Mean Annual rates per million. Median Age of Notified Cases: England and Wales, 1946–49.

		All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown	Median Age
MALES											
Poliomyelitis	Number	8,143	332	1,311	1,138	1,810	1,153	1,193	1,152	54	8·49
	Per cent. distribution ...	100	4	16	14	22	14	15	14	1	
	Rate per million	103	209	437	427	309	205	130	22		
Polioencephalitis ...	Number	726	31	92	88	168	123	91	130	3	9·48
	Per cent. distribution ...	100	4	13	12	23	17	13	18	0	
	Rate per million	9·2	20	31	33	29	22	10	2·5		
FEMALES											
Poliomyelitis	Number	6,665	282	1,049	873	1,323	863	1,078	1,155	42	9·19
	Per cent. distribution ...	100	4	16	13	20	13	16	17	1	
	Rate per million	75	188	368	346	234	159	92	20		
Polioencephalitis ...	Number	514	24	48	64	86	59	86	145	2	12·88
	Per cent. distribution ...	100	5	9	13	17	11	17	28	0	
	Rate per million	5·8	16	17	25	15	11	7·3	2·4		

There was a male excess in both forms of the disease at each age but this male excess was notably large in respect of the encephalitic form of the

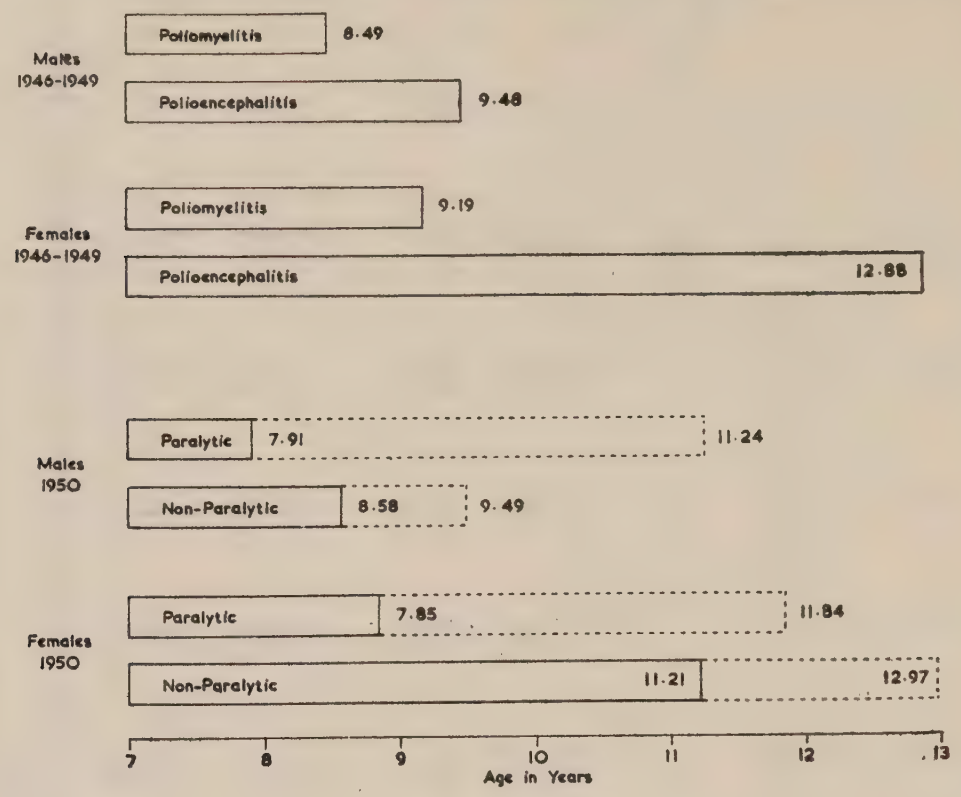


FIG. 6

Median age of notified cases of Poliomyelitis and of Polioencephalitis, 1946–49; and of Paralytic and Non-Paralytic Poliomyelitis, 1950. England and Wales. The extensions in dotted lines represent the median age of paralytic and non-paralytic cases at ages 3 and over.

disease at ages 1–2 and 5–14. At other ages there was little difference between the sex ratios of poliomyelitis and of polioencephalitis.

Paralytic and Non-Paralytic Poliomyelitis

(*Table* 10)

Since the beginning of 1950 poliomyelitis has been notifiable either as paralytic or non-paralytic. During 1950, 5,555 cases were notified as paralytic, and 2,195 as non-paralytic, the relative percentages of the two forms at each age being:—

	All ages	0–	1–	3–	5–	10–	15–	25+
Male:—								
Paralytic	70	84	82	67	61	59	71	78
Non-paralytic ...	30	16	18	33	39	41	29	22
Female:—								
Paralytic	74	92	85	76	71	64	69	72
Non-paralytic ...	26	8	15	24	29	36	31	28

At ages under 15 the percentage notified as paralytic was higher among female cases than male but at higher ages this relationship was reversed.

In each sex the percentage paralytic was highest in infancy possibly due to failure to recognise non-paralytic cases at early ages, declined during child-

TABLE 10

Notifications of Paralytic and of Non-Paralytic Poliomyelitis. Numbers, Percentage Age Distribution, Rate per million. Median Age of Notified Cases. England and Wales, 1950.

		All Ages	0–	1–	3–	5–	10–	15–	25 and over	Unknown	Median Age
MALES											
Paralytic	Number	2,943	137	537	425	629	298	395	509	13	7·91
	Per cent. distribution ...	100	5	19	14	22	10	13	17	0	
	Rate per million	139	384	699	544	400	207	140	38		
Non-paralytic ...	Number	1,276	26	119	205	398	210	165	147	6	8·58
	Per cent. distribution ...	100	2	9	16	32	16	13	12	0	
	Rate per million	61	73	155	263	254	146	58	11		
FEMALES											
Paralytic	Number	2,612	119	500	396	501	257	372	457	10	7·85
	Per cent. distribution ...	100	5	19	15	20	10	14	17	0	
	Rate per million	115	350	680	533	334	185	128	30		
Non-paralytic ...	Number	919	10	91	123	200	143	169	181	2	11·21
	Per cent. distribution ...	100	1	10	13	22	16	18	20	0	
	Rate per million	40	29	124	166	133	103	58	12		

hood but tended to rise again at adult ages. As a result the median age of non-paralytic cases in each sex was higher than of paralytic cases (Figure 6) and 38 per cent. of male and 39 per cent. of female paralytic cases were at ages under 5, compared with 27 per cent. of male and 24 per cent. of female non-paralytic cases at these ages.

If notified cases at ages under 3 years are excluded to avoid possible distortion from defective notification of non-paralytic cases at young ages, the age-relationship between the two forms of the disease is considerably altered. The median age of notified cases in each sex at ages 3 and over becomes:—

	Paralytic	Non-paralytic
Males	11·24	9·50
Females	11·85	12·97

The sex ratios at each age (male notification rates per cent. of female) were as follows:—

	All ages	0–	1–	3–	5–	10–	15–	25+
Paralytic...	121	110	103	102	120	112	109	127
Non-paralytic	153	252	125	158	191	142	100	92

In both types of the disease the total male rates exceeded the female but whereas the male excess of paralytic cases was fairly constant from age to age, ranging between a 2 per cent. to a 27 per cent. male excess, the sex ratio of non-paralytic cases showed much wider age differences, varying from a 152 per cent. excess of male cases in infancy and a 91 per cent. male excess at 5–9 to an 8 per cent. deficit of male cases at ages 25 and over. The high sex ratio of non-paralytic cases at childhood ages could possibly be explained by a failure to recognise and to notify cases of non-paralytic poliomyelitis in girls as readily as in boys. There is, however, no obvious reason why such a failure of notification of female cases should occur and Benjamin and Taylor (1951) examining the phenomena in hospital cases in 1947 and 1949 were unable to find evidence in support of the suggestion of defective notification of female cases.

Notifications at ages under one year

(*Table* 11)

In view of the evidence of relationship between inoculations and poliomyelitis and the suggestion (Bousfield, 1951) that if children received their immunisations early in infancy they would run much less risk of poliomyelitis it is of some interest to study the distribution of notified cases during the first year of life. Ordinarily no details are obtained centrally about the precise age of notified cases under one year, but for 1950 this information was specially obtained from Medical Officers of Health and is shown in Table 11 and Figure 7, without distinction of sex.

TABLE 11

Paralytic and Non-Paralytic Poliomyelitis. Notifications at ages under one year. England and Wales, 1950

	Total under one year	Age in Months											Unknown (but under one year)
		0–	1–	2–	3–	4–	5–	6–	7–	8–	9–	10–	11–	
Paralytic	254	5	7	14	12	15	19	25	21	27	28	43	34	4
Non-paralytic	37	—	—	2	—	2	6	5	5	7	4	4	2	—
TOTAL	291	5	7	16	12	17	25	30	26	34	32	47	36	4

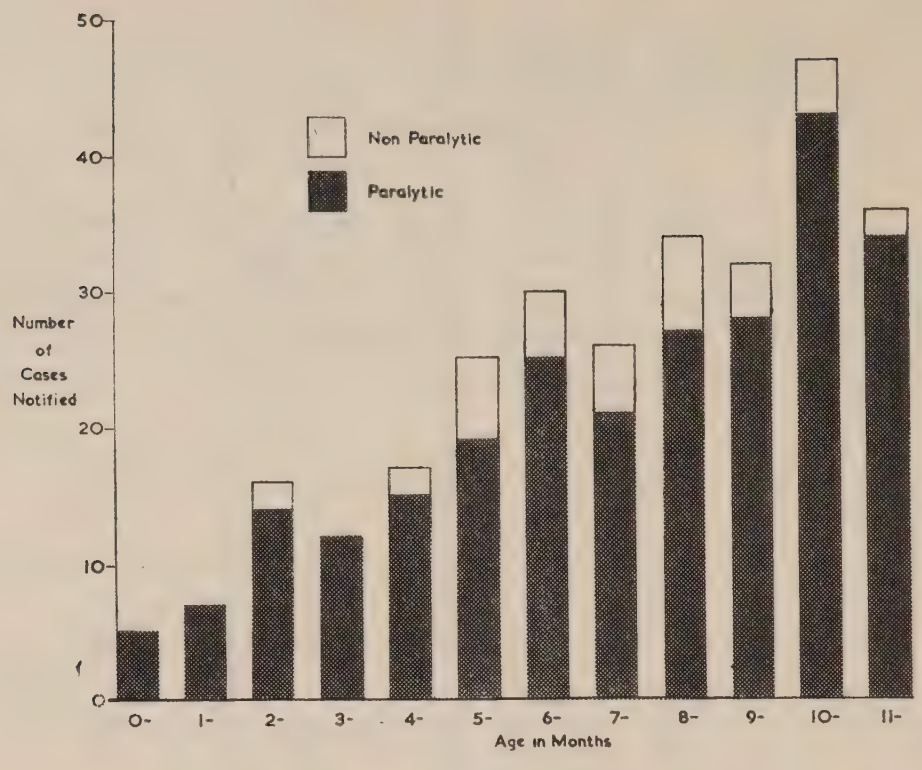


FIG. 7

Poliomyelitis, Paralytic and Non-Paralytic. Distribution of notified cases at ages under one year. England and Wales, 1950.

Combining some of the separate months of age, the distribution of cases reported as paralytic or as non-paralytic was as follows:—

Age	Paralytic	Non-paralytic	Paralytic per cent. of total
Under 1 month	5	—	100
1 and under 3 months	21	2	91
3 and under 6 months	46	8	85
6 and under 9 months	73	17	82
9 and under 12 months	105	10	90
Not stated	4	—	100
Total under 1 year	254	37	87

Of the paralytic cases 10 per cent. were at ages under 3 months and a further 18 were aged 3 to 6 months. Only 5 per cent. of non-paralytic cases were aged under 3 months, and none was under one month. Commenting on the incidence of poliomyelitis in infancy MacNalty (1936) said:—"It is not altogether certain whether the disease occurs *in utero*: Morton, Batten and others have reported cases which they considered examples of intrauterine infection. The disease not infrequently occurs within the first years of life; 23 of the 228 cases notified in England and Wales in 1918 fell within this age-period. Duchenne reported a case twelve days after birth, Bramwell one of 3 weeks of age, and Sinkler two cases under 1 month of age."

Wright and Owen (1952) have reported a case developing at the eighth day of life in a child whose mother had developed poliomyelitis the day before delivery. Commenting upon the rarity of the disease during the first month they remarked:—"Severin (1939), in a review of the literature, was able to find only twenty-two cases that occurred in the first month of life, while Baskin *et al* (1950) could find only ten cases in the first fortnight, which they consider the newborn period. They reported two further cases; in one the illness began on the fourth day of life and death occurred on the seventh, and in the other the illness began on the ninth day and death occurred on the fifteenth."

TABLE 12

Fatal Poliomyelitis. Interval between onset of disease and death. England and Wales, 1950 *(excluding cases without statement of interval).*

	Under 1 week	1 week –1 month	1 and under 3 months	3 and under 6 months	6 and under 9 months	9 and under 12 months	Total under 12 months
A.—INTERVAL LESS THAN ONE YEAR							
Ages:							
0–4	9	6	—	2	—	—	17
5–14	6	7	2	4	2	—	21
15–44	35	13	1	6	—	1	56
45 and over	3	1	—	—	—	—	4
TOTAL—All Ages	53	27	3	12	2	1	98
Males	*31*	*14*	*2*	*5*	*1*	*1*	*54*
Females	*22*	*13*	*1*	*7*	*1*	—	*44*
Poliomyelitis	*33*	*21*	*2*	*12*	*2*	—	*70*
Polioencephalitis	*20*	*6*	*1*	—	—	*1*	*28*
Years	1–	2–	3–10	11–20	21–50	51 or over	Total over 1 year
B.—INTERVAL MORE THAN ONE YEAR							
Ages:							
0–4	—	—	—	—	—	—	—
5–14	1	1	—	—	—	—	2
15–44	4	1	—	2	1	—	8
45 and over	—	—	—	—	2	1	3
TOTAL—All Ages	5	2	—	2	3	1	13
Males	*2*	*1*	—	—	*2*	*1*	*6*
Females	*3*	*1*	—	*2*	*1*	—	*7*

Fatal Poliomyelitis : Interval between onset and death

(*Table* 12)

The new International Form of Death Certificate introduced in 1950 has reintroduced a panel for the statement of the interval between the onset of each disease mentioned and death. Details of interval between onset of poliomyelitis and death were given on 111 death certificates in 1951. In 98 cases the duration of survival was less than one year and in 53 cases less than one week. The longest interval recorded was 67 years.

Summary

Poliomyelitis attained exceptional epidemic prevalence in England and Wales in 1947, 1949 and 1950.

At each age notified cases and deaths were more numerous among males than females. The notification rate was highest at ages 1 and 2, the death rates highest under 1 year, and fatality rates highest at adult ages.

Notification and death rates at ages under 5 were higher in urban than rural districts, but this relationship was reversed at higher ages.

Three geographical regions had the highest notification rates in one of the three epidemic years, and the lowest rate in another of these three years.

During 1944–50 the median age of notified cases tended to be highest in the first and lowest in the second quarter of the year. The median age of male cases in 1944–50 was 8·46 years and of female cases 9·15 years. The corresponding median age of male notified cases in 1912–19 was 3·93 years and of female cases 3·90 years.

The median age of notified cases was highest in the geographical regions that had the lowest infant mortality rates. The median age of notified cases was lower in the large towns than in the rural districts.

The median age of cases notified in 1946–49 as poliencephalitis, especially female cases, was higher than of those notified as poliomyelitis.

The median age of cases notified as non-paralytic in 1950 was higher than of paralytic cases.

254 paralytic and 37 non-paralytic cases were notified at ages under 1 year, and of these 26 paralytic and 2 non-paralytic cases were aged under 3 months.

Out of 111 fatal cases about which information was obtained on duration of survival, 53 died within one week of the onset of the disease.

References

Baskin, J. L., Soule, E. H., and Mills, S. D. (1950) *Am. J. Dis. Child.*, **80**, 10 (Quoted by Wright and Owen).

Batten, F. E. (1910) *Brain*, XXXIII, 149 (Quoted by MacNalty).

Benjamin, B. and Breen, G. E. (1950) *B.M.J.*, ii, 1473.

Benjamin, B. and Gale, A. H. (1949) *Monthly Bull., Min. of Health and P.H.L.S.*, October, p. 208.

Benjamin, B. and Taylor, I. (1951) *Lancet*, ii, 689.

Bousfield, G. (1951) *Lancet*, i, 1029.

Bramwell, B. (1908) *Studies in Clinical Medicine*, No. 1, i, 11 (Quoted by MacNalty).

Buzzard, T. (1898) *Lancet*, i, 847.

Duchenne, G. B. A. *L'Electrisation Localisée*, 3rd Ed., p. 381 (Quoted by MacNalty).

Gale, A. H. (1948) *Monthly Bull., Min. of Health and P.H.L.S.*, June, p. 127.
Gale, A. H. (1949) *Proc. Roy. Soc. Med.* (*Epid. & Soc. Med*), XLII, 47.
Gale, A. H. (1951 *a*) *B.M.J.*, i, 1511.
Gale, A. H. (1951 *b*) *The Medical Press*, July 25th, p. 105.
Gunn, W. (1948) *Proc. of B.M.A. Annual Meeting*, p. 197, Butterworth & Co.
Local Government Board. (1911–12) *Report of Medical Officer*, p. xxxii, London, H.M.S.O.
Low, R. B. (1915–16) Appendix 4 to Report of Medical Officer of Local Government Board, London, H.M.S.O.
MacEwen, H. A. (1912) Report to Local Government Board on Public Health and Medical Subjects, New Series No. 61, p. 138, London, H.M.S.O. (Also published as Appendix 4 of Report of Medical Officer, Local Government Board, 1911–12, p. 54).
MacNalty, A. S. (1936) *B.M.J.*, ii, 57.
Martin, W. J. (1951) *Brit. J. Soc. Med.*, **5**, 236.
Morton. (1890) *Philadelphia Med. News*, July 12th (Quoted by MacNalty).
Pasteur, W. (1896–97) *Trans. Chir. Soc., London*, **30**, 143 (Quoted by Gale, 1951 *b*).
Rhodes, A. J. (1947) *Bull. Hygiene*, **22**, 353.
Richmond, A. E. (1951) *J. Roy. Army Med. Corps*, **97**, 15.
Severin, G. (1939) *Nord. Med.* (*Hygeia*), i, 55 (Quoted by Wright and Owen).
Sinkler. (1875) *Am. J. Med. Sc.*, April (Quoted by MacNalty).
Sutherland, I. N. (1951) *Health Bulletin of Department of Health for Scotland*, IX, **1**, 13.
Weinstein, L., Shelokov, A., Seltser, R., and Winchell, G. D. (1952) *New England J.M.* 246, 8, 296.
Wright, G. A. and Owen, T. K. (1952) *B.M.J.*, i, 800.

SURVEY OF SICKNESS—DECEMBER QUARTER, 1951

Issued from the General Register Office, Somerset House, W.C.2

This note relates to sickness experience reported by random samples of the adult population at interviews conducted by the Social Survey on behalf of the Registrar General up to February, 1952. As an economy measure interviews for the Survey of Sickness have been suspended for the time being, and the present Report is the last in the continuous series which has appeared in this Bulletin from time to time since March, 1944.*

During the years in which the Survey of Sickness has been in operation reports and articles have also appeared in the Annual Report of the Chief Medical Officer of the Ministry of Health, in the Registrar General's Statistical Review (Text) and in various other publications.†

Results of the Social Survey's interviews carried out in November and December, 1951 and January and February, 1952 when the experience of October to December, 1951 was recorded are to be found in the Registrar General's Quarterly Return No. 413 (Tables A to H). The report given here presents some rates derived from these tables without standardisation or correction for the number of days in the month and compares them with those for preceding months.

Since the beginning of 1951, results from the Survey of Sickness have related only to persons aged 21 and over, and not 16 and over as formerly.

The "Monthly Health Index"—the proportion of persons per 100 interviewed who suffered no illness or injury, so far as they remembered, during the stated months—is given in Table A, separately for men and

* *This Bulletin:* 1944, **3**, pp. 46, 70, 93, 194; 1945, **4**, pp. 30, 80, 119, 198, 244; 1946, **5**, pp. 60, 131, 201; 1947, **6**, pp. 4, 123, 194; 1948, **7**, pp, 11, 79, 169, 213; 1949, **8**, pp. 9, 78, 148, 232; 1950, **9**, pp. 13, 89, 160, 221; 1951, **10**, pp. 11, 81, 162, 239, also p. 136; 1952, **11**, pp. 11, 86.

† Slater, P. (1946) "*Survey of Sickness, October, 1943 to December, 1945*". The Social Survey.
Stocks, P. (1949) "*Sickness in the Population of England and Wales 1944–47*". H.M.S.O.
Logan, W. P. D. (1950) *Lancet*, p. 773.
Brooke, Eileen M. (1951) *Lancet*, p. 1272.

women, at ages 21–64 and ages 65 and over. The Index was lower for men and higher for women during the December Quarter, 1951 than for the corresponding months in 1949 and 1950.

Table B gives monthly illness rates, the proportion of persons per 100 interviewed who suffered at least one illness during the month, excluding those with injury only, distinguishing persons with illness commencing in the month from those with illness continued from the previous month. The table also gives days of incapacity reported in each month and numbers of medical consultations. Including persons with an injury, the average rates for the December Quarter of 1949, 1950 and 1951 compare thus:—

	Ages 21–64			Ages 65 and over		
	Monthly Sickness	Incapacity	Medical Consultations	Monthly Sickness	Incapacity	Medical Consultations
Oct.–Dec., 1949 ...	68·8	99	42·0	82·5	267	66·1
Oct.–Dec., 1950 ...	69·6	98	43·0	84·5	179	68·1
Oct.–Dec., 1951 ...	69·0	94	41·0	85·2	135	65·5
Oct.–Dec., 1951 per cent. of Oct.–Dec.,						
1949	100	95	98	103	51	99
1950	99	96	95	101	75	96

The level of sickness during the three December Quarters remained practically unchanged, but the number of medical consultations fell slightly for both groups of ages. At ages 21–64 the level of incapacity also fell slightly, but at ages 65 and over this rate fell considerably, and was only half the level experienced in the December Quarter, 1949.

Table B also shows that there was an increase in the December over the September Quarter, 1951 in new illnesses, days of incapacity and medical consultations. The number of persons suffering from chronic illnesses showed a slight decrease because persons experiencing both a new and chronic illness are excluded from the latter category.

Table C distinguishes persons who began to suffer from a serious, moderate or mild illness during each month from those who developed an illness of a minor or ill-defined nature. Average monthly rates during the December Quarters of 1949, 1950 and 1951 compare thus:—

	Serious, Moderate, or Mild				Minor or Ill-Defined			
	Ages 21–64		65 and over		Ages 21–64		65 and over	
	M	F	M	F	M	F	M	F
Oct.–Dec., 1949	4·3	6·0	6·5	8·6	38·6	45·0	32·7	37·3
Oct.–Dec., 1950	7·2	8·5	10·1	12·6	37·1	43·5	31·7	36·5
Oct.–Dec., 1951	9·2	10·9	11·4	14·2	33·1	38·6	31·7	33·8

The rates of more serious illnesses were higher in the December Quarter of 1951 than in the previous year which in turn were an increase over 1949.

The rates of minor illnesses, however, continued generally to fall from the levels experienced in the preceding years ; the rate for men aged 65 and over showed only a small reduction since 1949.

Table D gives average monthly numbers of new illnesses from three selected causes (not persons ill as in Tables B and C) experienced in successive quarters by 100 persons of each sex and age, and the number of days of incapacity arising from each cause (including both new and continued illness). In this table the rates for the younger group relate to ages 16–64 in 1949–50, and to ages 21–64 in 1951, so that the rates shown for 1949 and 1950 would need to be increased by a small fraction to be properly comparable with those for 1951 (see this Bulletin, October, 1951, p. 241).

The incidence of influenza and colds during the December Quarter was well below that experienced in the corresponding quarters of 1949 and 1950. Incapacity due to this cause was also low compared with the same period in the two previous years. The incidence of other respiratory diseases showed, however, an increase over previous years and was the highest recorded for any quarter since the beginning of 1949 ; but as weather was mild there was no exceptional increase in days of incapacity.

Except for women aged 65 and over, the incidence of rheumatism was slightly higher than in the December Quarters of 1949 and 1950, and incapacity due to this cause was also a little higher than in the corresponding period of 1950. Incapacity rates for the older women were considerably lower than in 1950 and for both sexes, at ages 65 and over, the incapacity rate was greatly reduced from the high levels experienced by these groups in the December Quarter of 1949.

Days of incapacity from all causes per 100 persons interviewed by sex and age are shown below.

	Ages 21–64		Ages 65 and over	
	Males	Females	Males	Females
1949				
January–March	122	127	200	220
April–June	86	75	151	165
July–September	82	70	89	99
October–December ...	98	99	225	302
1950				
January–March	114	117	196	231
April–June	89	76	143	133
July–September	79	62	118	87
October–December ...	99	96	182	176
1951				
January–March	167	177	295	271
April–June	86	68	126	112
July–September*	80	58	88	73
October–December ...	99	90	122	143

* Corrected for absence of October interviews.

The incapacity rates for the December Quarter, 1951 were generally lower than in the two preceding years. For persons aged 65 and over there was a considerable reduction and, as noted above, the rate for 1951 was about half that experienced in the December Quarter of 1949.

A.—*Monthly Health Index: The proportion of persons, per hundred persons interviewed, for each sex and age, who reported having had no illness or injury during the month stated.*

Month of Experience	Ages 21–64						Ages 65 and over					
	M			F			M			F		
	1949	1950	1951	1949	1950	1951	1949	1950	1951	1949	1950	1951
January	35	33	28	25	25	21	20	21	15	12	12	8
February... ...	34	33	31	23	26	24	18	20	16	9	11	10
March	34	37	33	23	27	26	16	20	14	12	11	12
April	39	37	36	26	29	29	19	18	20	12	11	12
May	39	41	40	28	28	30	23	21	23	15	12	12
June	42	42	41	30	30	32	24	21	23	14	12	13
July	43	40	41	31	31	31	26	22	21	14	12	15
August	43	42	41*	31	31	31*	26	25	24*	16	11	16*
September ...	44	39	35*	31	29	29*	25	20	18*	15	13	11*
October	39	36	34	26	25	28	24	22	19	14	11	12
November ...	35	37	35	26	27	29	23	22	18	13	11	13
December ...	36	34	35	27	26	26	27	20	17	14	12	13

(Based on combined results of interviews in two following months.)

* Experience based on one month's interviews corrected by factors based on previous experiences.

B.—*Monthly Illness Rates with Days of Incapacity and Numbers of Medical Consultations, per* 100 *Persons.*

Month of Experience	Ages 21–64				Ages 65 and over			
	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month	With a new or recurrent illness starting	With illness continued from previous month	Days of incapacity during month	Number of medical consultations in month
1949								
January ...	44·4	25·2	112	48	44·2	39·7	217	72
February ...	46·9	24·7	132	51	44·7	41·3	207	71
March ...	45·9	25·7	131	47	43·7	42·1	211	83
April	39·7	27·9	87	41	38·5	46·5	175	74
May	38·3	28·0	76	41	33·1	48·1	163	61
June ...	34·5	28·9	78	39	32·6	47·8	142	60
July	33·5	29·4	79	42	33·1	46·6	118	63
August ...	32·8	29·5	72	38	32·7	46·9	71	54
September ...	36·1	26·1	75	39	35·9	44·6	96	60
October ...	44·0	23·3	94	43	40·6	40·9	212	59
November ...	47·0	21·7	105	42	42·8	39·4	293	73
December ...	50·9	20·5	102	42	46·3	36·5	323	65
1950								
January ...	43·9	23·2	115	43	42·2	39·7	212	68
February ...	47·1	22·9	119	51	43·9	40·3	211	77
March ...	44·8	22·9	111	50	42·2	43·3	221	87
April	41·7	24·7	87	42	42·6	43·5	183	85
May	41·2	24·2	80	43	40·5	43·9	133	80
June	37·6	25·9	80	43	36·9	47·0	94	64
July	38·2	25·4	71	38	39·5	44·1	76	65
August ...	38·4	24·3	64	37	37·5	44·9	104	62
September ...	42·5	23·3	75	41	39·8	44·1	120	73
October ...	47·6	21·7	85	44	44·8	39·5	137	61
November ...	47·3	20·6	90	43	45·5	38·4	170	70
December ...	50·4	19·4	117	42	48·0	35·7	227	73
1951								
January ...	56·5	18·5	233	60	54·9	33·7	371	88
February ...	49·7	22·0	166	54	47·4	39·5	271	87
March ...	47·9	22·6	118	49	45·7	41·5	202	80
April	43·0	24·1	86	45	41·1	43·2	153	74
May	40·7	24·1	79	42	42·3	40·6	112	70
June	40·1	23·0	66	39	42·4	40·2	87	65
July	39·9	23·4	67	38	41·2	41·0	75	63
August* ...	41·7	24·2	64	37	42·3	38·6	64	53
September* ...	37·4	25·9	71	40	38·3	44·4	96	63
October ...	45·3	23·1	86	42	43·7	41·2	113	65
November ...	43·6	22·6	96	40	44·9	39·7	135	67
December ...	49·5	20·0	101	40	49·2	36·2	155	65

Notes.—People who experienced both a continued illness and a new or recurrent one are included in the rate for new and recurrent. Persons with an injury but no illness are excluded from both rates; but days of incapacity and medical consultations include those due to injury. (For numbers who suffered an injury, see Registrar General's Quarterly Returns Table E.)

* Uncorrected rates based on one month's experience.

C.—*Rates of Morbidity per* 100 *Males and Females for Illnesses starting in each Month, distinguishing Minor and Ill-defined complaints.*

Month of Experience	Serious, Moderate or Mild				Minor or Ill-defined			
	Ages 21–64		Ages 65 and over		Ages 21–64		Ages 65 and over	
	M	F	M	F	M	F	M	F
1949								
January ...	5·2	5·7	7·6	9·6	35·2	42·2	31·9	38·1
February ...	6·5	8·1	6·8	10·9	37·3	41·5	31·6	38·5
March ...	5·8	7·8	7·9	11·7	35·5	41·8	34·3	33·0
April	3·6	4·7	6·2	6·4	30·1	40·0	30·3	33·4
May	3·4	3·8	2·9	3·9	29·5	39·1	28·7	30·3
June	2·6	3·6	3·0	6·3	26·2	35·7	24·9	29·9
July	2·7	3·8	3·7	6·9	25·6	33·9	26·2	28·5
August ...	2·7	3·6	4·4	4·0	25·4	33·1	27·8	29·1
September ...	3·2	4·2	2·7	4·8	28·1	35·8	31·5	32·3
October ...	3·6	5·8	5·6	6·3	35·3	42·4	33·1	35·6
November ...	4·8	5·4	8·5	9·7	38·1	45·0	32·7	34·1
December ...	4·5	6·8	5·4	9·7	42·3	47·7	32·4	42·2
1950								
January ...	3·7	5·8	9·6	8·0	35·5	42·0	28·0	37·2
February ...	6·0	8·0	9·2	9·2	36·5	43·0	31·4	37·0
March ...	6·1	8·1	8·4	11·3	32·6	42·0	29·4	33·8
April	5·4	5·6	5·5	10·6	33·3	38·8	33·7	34·4
May	4·6	4·9	5·5	6·6	31·8	40·4	29·8	37·8
June	3·2	4·1	3·6	5·7	28·3	38·8	27·9	34·9
July	3·3	4·5	6·1	6·6	30·5	37·6	27·0	37·6
August ...	3·2	4·3	4·6	5·7	29·7	38·9	26·2	36·9
September ...	4·3	4·8	5·5	7·0	34·1	41·3	31·0	35·0
October ...	5·5	7·0	8·6	10·0	38·3	43·8	32·5	37·1
November ...	6·6	7·6	9·7	10·7	36·0	43·8	32·9	36·7
December ...	9·5	10·9	12·1	17·0	37·0	42·8	29·8	35·8
1951								
January ...	15·6	19·5	18·2	27·2	38·0	39·6	31·2	31·9
February ...	10·4	12·5	13·5	16·2	35·2	40·8	31·6	33·0
March ...	7·1	8·8	11·3	13·2	36·9	42·4	32·3	34·0
April	4·8	6·1	8·4	10·9	34·0	40·6	29·7	32·3
May	4·6	5·4	6·5	8·9	30·9	39·9	33·8	34·9
June	3·9	4·6	5·0	8·5	32·0	39·0	34·4	36·2
July	3·7	4·7	5·7	7·3	31·4	39·4	33·5	35·1
August* ...	4·3	4·1	5·0	8·0	32·6	41·9	31·7	38·0
September* ...	5·5	6·7	4·5	12·1	27·2	34·6	23·4	33·3
October ...	8·5	10·9	9·8	13·5	33·2	37·4	29·1	33·7
November ...	8·5	8·6	11·3	13·5	31·0	38·7	32·8	32·0
December ...	10·6	13·1	13·0	15·5	35·1	39·7	33·3	35·7

Notes.—For definition of categories see Bulletin of April, 1944. Only the illness of highest category is taken account of when more than one occurred in a month, and injuries are excluded. " Ill-defined " excludes all symptomatic illness which caused incapacity for Work, such cases being classed to the appropriate higher category. " Illnesses starting in each month " include new and recurrent illnesses.

* Uncorrected rates based on one month's experience.

D.—*Average Monthly Incidence of Certain Types of Illness and Average Days of Incapacity.*

Period of Experience	Number of new illnesses and injuries (or attacks of old ones) in a month per 100 people				Average days of incapacity in a month per 100 people			
	Ages 21–64*		65 and over		Ages 21–64*		65 and over	
	M	F	M	F	M	F	M	F
	Influenza and Colds							
1949								
January–March ...	21·6	21·4	15·9	18·3	32·1	39·1	36·4	62·5
April–June	9·1	9·9	7·2	6·2	8·7	9·0	17·7	14·0
July–September ...	6·6	6·3	4·0	4·5	3·5	4·9	3·7	4·0
October-December ...	21·3	20·9	12·3	16·0	13·3	17·8	9·7	23·7
1950								
January–March ...	18·4	18·0	12·0	13·5	26·3	28·8	27·4	40·5
April–June	9·3	8·5	7·0	6·6	10·1	9·7	17·3	19·9
July–September ...	8·7	8·3	5·9	6·3	4·3	6·0	2·1	8·1
October–December ...	19·3	19·5	13·9	13·7	15·7	22·3	36·8	31·6
1951								
January–March ...	22·9	22·5	17·2	18·0	67·3	81·4	78·3	103·2
April–June	7·6	6·5	6·2	4·6	5·6	5·3	9·4	5·9
July–September ...	5·4	4·9	3·1	2·7	2·4	3·4	0·2	4·3
October–December ...	12·9	12·2	8·8	7·9	9·9	14·6	14·2	13·1
	Other respiratory disease							
1949								
January–March ...	2·8	3·0	4·6	3·6	13·7	13·2	44·7	47·9
April–June	2·5	2·9	4·1	1·9	9·1	7·0	25·4	13·6
July–September ...	2·8	2·1	3·2	2·6	8·3	5·1	8·2	6·0
October–December ...	3·1	3·4	6·3	4·9	13·7	8·7	40·2	41·0
1950								
January–March ...	2·8	4·2	4·4	3·9	14·8	14·9	46·6	39·3
April–June	4·0	4·1	4·4	4·5	12·8	9·3	16·1	17·1
July–September ...	3·7	3·7	4·6	2·8	7·1	3·6	12·0	4·6
October–December ...	5·6	6·6	7·4	7·1	15·5	15·5	35·1	48·6
1951								
January–March ...	6·4	6·7	8·5	7·9	25·3	30·2	83·5	73·5
April–June	4·9	4·7	6·3	6·2	14·0	6·4	25·8	20·5
July–September ...	4·6	4·3	5·3	6·1	8·6	4·5	9·7	8·5
October–December ...	8·7	9·3	10·1	10·3	14·1	15·6	35·3	31·0
	Rheumatism, all forms							
1949								
January–March ...	5·3	7·3	10·7	11·1	8·5	7·7	10·7	21·1
April–June	4·5	6·8	7·7	9·3	5·3	5·0	13·1	34·6
July–September ...	4·3	5·8	9·5	8·0	6·1	5·1	9·2	17·4
October–December ...	5·2	7·6	7·6	9·2	6·4	8·6	30·7	54·7
1950								
January–March ...	4·9	8·2	10·2	10·1	7·8	8·0	15·6	30·5
April–June	5·3	7·8	8·9	10·3	5·9	5·8	9·2	22·2
July–September ...	5·1	8·0	7·9	11·3	4·3	4·3	10·3	12·6
October–December ...	6·5	9·1	9·3	13·1	4·7	5·8	6·3	16·9
1951								
January–March ...	8·2	10·5	10·4	14·5	10·9	7·3	18·6	18·0
April–June	6·3	9·1	11·3	15·0	4·8	5·2	8·5	11·0
July–September ...	6·3	8·6	8·5	14·0	6·8	6·2	7·7	5·4
October–December ...	6·8	9·6	9·7	11·6	6·6	7·1	9·8	8·4

Note: The days of incapacity are those caused by all illnesses of the nature specified regardless of when the illness began (i.e., new, recurrent, or continued from the previous month).

* Rates for 1949 and 1950 include persons aged 16–20.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, JUNE, 1952

Issued from the General Register Office, Somerset House, W.C.2

	June 7th	June 14th	June 21st	June 28th	Average weekly figures, June, 1951
Scarlet Fever	843	845	1,071	1,212	950
Whooping Cough	2,075	2,513	2,575	2,444	3,352
Diphtheria	29	19	23	33	36
Measles, excluding Rubella	6,348	7,312	6,288	6,843	12,792
Acute Pneumonia	371	427	384	310	417
Meningococcal Infection	23	40	33	30	38
Acute Poliomyelitis (Paralytic) ...	42	28	49	58	38
,, ,, (Non-paralytic) ...	19	20	19	32	28
Ophthalmia Neonatorum	36	31	38	36	33
Puerperal Pyrexia and Puerperal Sepsis	204	210	269	233	74
Dysentery	210	188	145	135	600
Paratyphoid	13	32	38	36	23
Typhoid	5	4	6	4	9
Smallpox	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1

Central Public Health Laboratory: Change of telephone number

From 1st July, 1952, the telephone number of the Central Public Health Laboratory, Colindale Avenue, London, N.W.9, will be Colindale 7041 (eight lines). The old numbers 6041 and 4081 will no longer be used.

British Standards Institution

Readers of the Bulletin may be interested to know that the Institution has recently published a new British Standard (B.S.611:1952), in which are specified the dimensions, material and finish of Petri dishes suitable for general medical and bacteriological use.

AN ECONOMICAL TECHNIQUE FOR STREPTOCOCCAL GROUPING

H. A. Tarr, Public Health Laboratory, Oxford

By the technique to be described grouping of streptococci may be carried out with the use of very small amounts of extract and serum. The apparatus consists of a Pasteur pipette made from tubing 6 mm. in external diameter. The capillary end, which is cut off about 35 mm. from the shoulder, should correspond approximately to hole No. 55 in a Starrett wire gauge. This tube will fit conveniently into the type of rack used for Dreyer's agglutination tubes. The end of the capillary tube must not come in contact with the rack.

The capillary is brought into contact with the streptococcal extract, which is allowed to rise by capillary attraction to a height of approximately 6 mm. (Fig. 1). The height is controlled by a finger on top of the tube; any excess of extract may be expelled by pressure with the finger while the capillary is touching the side of the tube.

The grouping serum is added with a sterile platinum loop of about 3 mm. diameter. A loopful of serum is applied to the end of the capillary tube; it rises up the tube by capillary attraction and comes into contact with the streptococcal extract (Fig. 2).

In positive reactions the ring of precipitate develops rapidly at the junction of the two fluids and is clearly visible.

One advantage of this technique is that it can be carried out with 0·007 ml. of extract, and the same quantity of serum, thus effecting a considerable economy in serum. Another advantage, particularly for laboratories testing only small numbers of strains, is that the serum is taken from the bottle with a loop instead of a pipette. Although some workers may prefer to discard the tubes after use, it is the practice in this laboratory to wash and use them again. This method of grouping has been in routine use for several months with satisfactory results. It may also be applied to other precipitation tests.

Summary

An economical method for serological grouping of streptococci in capillary tubes is described.

FIG. 1.—Capillary tube in Dreyer's tube just after contact with streptococcal extract.

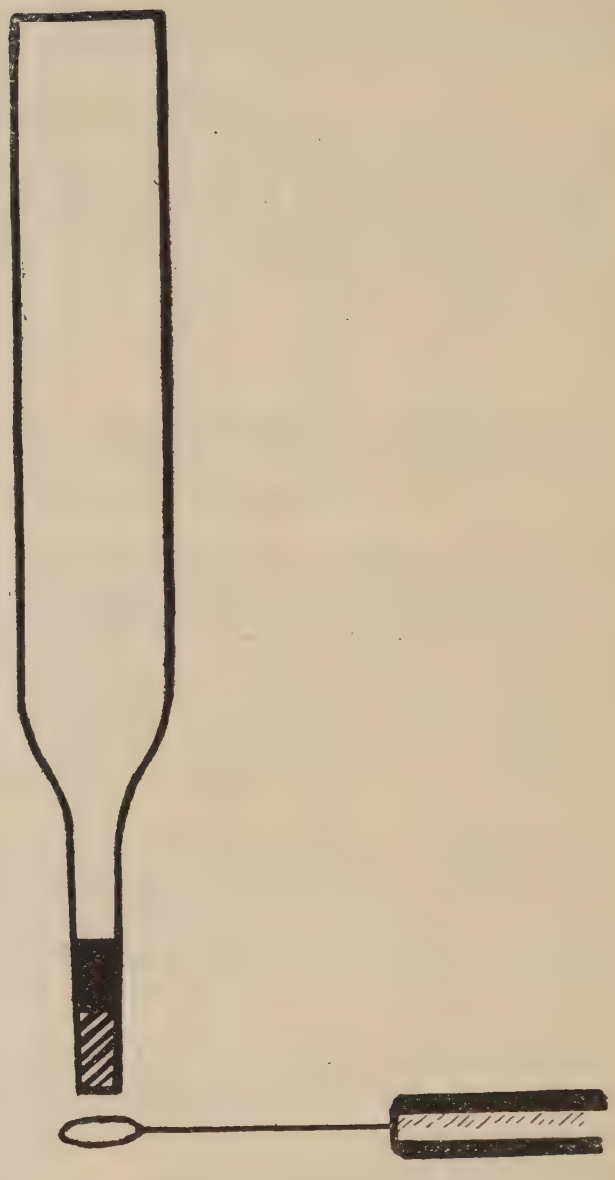


FIG. 2.—Method of applying loop containing serum to end of capillary tube containing streptococcal extract.

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.
Tel.: REGENT 8411. Extn. 91.
The June Bulletin was issued on 23rd June.

II.—LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(16122) Wt. 8221—7283 3300 7/52 D.L.

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

AUGUST 1952 Vol. 11

CONTENTS

Section I

Section II

Issued from the Office of the Ministry of Health, Savile Row, London, W.1.

POST-INFECTIOUS ENCEPHALITIS NOTIFIED IN 1951

E. T. Conybeare, O.B.E., M.D., F.R.C.P., Ministry of Health

During 1951 the number of cases of post-infectious encephalitis notified under the Public Health (Acute Poliomyelitis, Acute Encephalitis and Meningococcal Infections) Regulations, 1949, and recorded under category K in Table IV of the Registrar General's Weekly Returns for England and Wales was 119, this being a figure subject to revision as a result of the corrections* now submitted quarterly to the Registrar General.

In continuation of the enquiry begun in 1950, local health departments were asked to give in respect of each of these cases certain information supplementary to that already available from the Registrar General. Such information was received from medical officers of health regarding a total of 114 cases notified in 1951.

An analysis of this information shows the following points:—

1. Amended Diagnosis

(*a*) *Polioencephalitis.* In 4 of the notified cases the disease associated with the encephalitis was subsequently judged to be poliomyelitis and the notification was accordingly corrected.

(*b*) *Not Encephalitis.* In 19 of the notified cases the diagnosis was subsequently amended to one of some disease other than acute encephalitis and the notification was withdrawn.

2. Post-Infectious Encephalitis

In 91 of the notified cases it was found that the original diagnosis and inclusion in category K of Table IV of the Registrar General's Weekly return could be upheld. The infectious diseases associated with these 91 cases, the number of cases and the number of deaths is shown in the following table:—

Infectious Disease	(1) Notified Cases with Acute Encephalitis (Post-Infectious)	(2) Fatal Cases included in (1)
Measles	32	4
Mumps	37	0
Chicken Pox	9	0
Influenza	4	2
Vaccinia	2	0
Rubella	2	0
Herpes	2	0
Whooping Cough	2	0
Scarlet Fever	1	0
	91	6

* It was shown by the corrections to the 1950 figures and again in 1951 that there are also many changes of diagnosis from other diseases to acute encephalitis (post-infectious). No account of such changes has been taken in the above report.

If the above figures are compared with those resulting from a similar analysis of the information collected during 1950 (this Bulletin, March, 1951, p. 58) it will be seen that post-measles, encephalitis was notified twice as frequently during 1951 as in 1950 and post-mumps, encephalitis three times as frequently.

The rise during 1951 in the notifications of post-measles, encephalitis was commensurate with the rise in the notifications of measles which were nearly twice as numerous in 1951 as in 1950.

MINISTRY OF HOUSING AND LOCAL GOVERNMENT

While the Ministry of Housing and Local Government is now the central authority for housing, water-supplies, sewerage and sewage-disposal, river-pollution, public cleansing, and atmospheric pollution, medical advice on these subjects is still provided by the medical staff of the Ministry of Health. Medical Officers of Health who wish to communicate directly on any of these aspects of environmental hygiene should, therefore, write to the Chief Medical Officer, or to the Principal Medical Officer, Med. I, Ministry of Health, Savile Row, London, W.1.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, JULY, 1952

Issued from the General Register Office, Somerset House, W.C.2

	July 5th	July 12th	July 19th	July 26th	August 2nd	Average weekly figures, July, 1951
Scarlet Fever	1,036	1,038	1,038	1,173	997	802
Whooping Cough	2,347	2,243	2,206	2,040	2,043	3,319
Diphtheria	30	33	25	21	19	31
Measles, excluding Rubella ...	6,724	7,544	7,225	7,363	7,317	6,944
Acute Pneumonia	314	329	259	247	228	326
Meningococcal Infection ...	23	24	25	34	20	37
Acute Poliomyelitis (Paralytic)	61	78	105	157	139	50
,, ,, (Non-paralytic)	25	41	59	96	91	68
Ophthalmia Neonatorum ...	38	30	34	39	34	38
Puerperal Pyrexia and Puerperal Sepsis	234	276	213	260	227	92
Dysentery	180	151	140	135	93	297
Paratyphoid	21	25	39	75	130	36
Typhoid	2	3	2	5	5	6
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

VENEREAL DISEASES

Analysis of the total number of new patients attending the clinics in England and Wales during the quarter ending 31st March, 1952*

Number of patients* attending for the first time during the three months ending 31st March, 1952, and diagnosed as follows:	M	F	M	F	M	F	Totals
(*a*) Syphilis, primary ...	162	35					
(*b*) ,, secondary...	62	57	283	135			
(*c*) ,, latent in 1st year of infection	59	43					
(*d*) Syphilis, Cardio-vascular*	95	33					
(*e*) Syphilis of the nervous system†	165	83	665	600	1,037	882	1,919
(*f*) Syphilis, all other late or latent stages* ...	405	484					
(*g*) Syphilis, congenital (under 1 year) ...	14	18	89	147			
(*h*) Syphilis, congenital (over 1 year) ...	75	129					
(*i*) Gonorrhoea... ...	—	—	—	—	3,502	826	4,328
(*j*) Chancroid	—	—	—	—	102	2	104
(*k*) Lymphogranuloma venereum (Syn. Lymphogranuloma inguinale)... ...	—	—	—	—	16	1	17
(*l*) Granuloma inguinale (Syn. Granuloma venereum) ...	—	—	—	—	—	—	—
(*m*) Non-gonococeal urethritis (males only)	—	—	—	—	2,524	—	2,524
(*n*) Any other conditions requiring treatment	—	—	—	—	2,886	2,226	5,112
(*o*) Conditions not requiring treatment	—	—	—	—	6,722	3,190	9,912
(*p*) Conditions still remaining undiagnosed	—	—	—	—	231	188	419
	—	—	—	—	17,020	7,315	24,335

* Patients who have previously received treatment for the same condition at any Treatment Centre, or by a General Practitioner approved under Ministry of Health Circular 2226 are excluded.

† In order to avoid duplication, patients with cardio-vascular syphilis who are also suffering from syphilis of the nervous and/or other systems are recorded as suffering from cardio-vascular syphilis alone.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1

Hull Laboratory

Dr. J. H. McCoy has been appointed Director of the laboratory in succession to Dr. C. L. Greening, who resigned his post at the end of May to take up an appointment in Northern Rhodesia.

Leicester Laboratory

Dr. N. S. Mair has been appointed Director of the laboratory as from 1st July, 1952.

CULTIVATION OF MYCOBACTERIUM TUBERCULOSIS
PART I.—METHODS OF HOMOGENIZATION

A Report by a Working Party of the Public Health Laboratory Service on the Laboratory Diagnosis of Tuberculosis.*

INTRODUCTION

The demonstration of acid-fast bacilli in sputum, or any other material derived from the lower part of the respiratory tract, is commonly accepted as laboratory confirmation of suspected pulmonary tuberculosis. Unless the organisms are present in considerable numbers microscopic examination of stained films often fails to reveal them and in these circumstances it is necessary to resort to cultural methods or to animal inoculation. In recent years it has become essential to isolate the organism in order to test its sensitivity to drugs or antibiotics.

Some form of pre-treatment of sputum is necessary to homogenize the specimen and to inhibit contaminating organisms. This treatment is based on the fact that acid-fast bacilli are relatively more resistant in the cold than other organisms to the lethal effects of certain chemicals. Several chemical agents have been employed for this purpose, but many of the reports on their use have been contradictory.

ILLUSTRATIVE LITERATURE

Griffith (1914) used antiformin to destroy contaminating organisms. Petroff (1915) used 3 per cent. NaOH and his method was found to be superior to antiformin by Lurie (1923).

Löwenstein (1924) used 6 per cent. H_2SO_4 and 3 per cent. HCl. Sweany and Evanoff (1928) and Madsen (1950) found NaOH superior to acid, but Corper and Uyei (1927) found 6 per cent. H_2SO_4 superior to NaOH.

Jungmann (1938) introduced an iron-acid peroxide method and this was reported on favourably by Nassau (1942).

Corper and Stoner (1946) used 23 per cent. trisodium phosphate and found it superior to 5 per cent. oxalic acid and 3 per cent. NaOH. Starkey and Aubert (1950) and Gifford *et al.* (1951) found trisodium phosphate better than NaOH and van Vranken (1947) found it superior to oxalic acid. Byham (1950), however, found NaOH, and Beattie (1949) found 3 per cent. HCl superior to trisodium phosphate.

Mullahy (1950), using NaOH and trisodium phosphate, found that these two substances gave approximately the same incidence of positive cultures, but trisodium phosphate gave the higher contamination rate. Spendlove *et al.* (1949), using a number of substances, found NaOH and trisodium phosphate the least toxic to tubercle bacilli.

---

* Dr. G. T. Cook (Oxford), Dr. J. M. Croll (Lincoln), Dr. C. C. B. Gilmour (Peterborough), Dr. C. L. Greening (Secretary) (Hull), Dr. H. D. Holt (Colindale), Dr. J. E. Jameson (Brighton), Dr. R. Knox (Oxford), Dr. J. McCoy (Cambridge), Dr. J. Marks (Cardiff), Dr. P. H. Martin (Ipswich), Dr. A. I. Messer (Newcastle-on-Tyne), Dr. R. N. Phease (Convener) (Stafford), Dr. J. M. Ritchie (Birkenhead) and Dr. R. L. Vollum (Oxford).

Weed *et al.* (1951) in a general review of laboratory methods considered sodium hydroxide the best substance for concentrating smears and preparing sputum for culture. For specimens examined within a few hours of collection, they recommended the addition of an equal quantity of 1·5 per cent. NaOH, followed by shaking in a Kahn shaker for 30 minutes at room temperature. Trisodium phosphate was recommended for adding to sputum to be sent through the post, to prevent gross contamination.

Baker (1951) found sodium hydroxide superior to Jungmann's method, trisodium phosphate, or oxalic acid for culture, but the latter two methods were better for concentrating sputum.

Rasmussen (1951) found that sodium hydroxide was five times as lethal to tubercle bacill as sulphuric acid.

This diversity of opinion is difficult to explain, but the results obtained by different workers at different times and places are not easy to compare since it cannot always be assumed that the techniques employed were identical. It therefore seemed desirable to re-examine the merits of different homogenization methods under strictly comparable conditions. In order that the experiment should be on a sufficiently large scale, it was necessary for a number of laboratories to participate, but since all were constituent units of the Public Health Laboratory Service it was possible to ensure that like procedures were followed in every controllable detail.

The following report describes an investigation on these lines which was carried out in 1949–51.

GENERAL PLAN OF INVESTIGATION

Before undertaking laboratory investigations the advice of Professor A. Bradford Hill, Dr. J. O. Irwin and Dr. P. Armitage, of the Medical Research Council's statistical unit, was sought on the correct statistical approach, and the experimental design was based on their recommendation.

Three main factors to be considered were:—

(*a*) Methods of homogenization;

(*b*) The composition of the series of specimens;

(*c*) Uniformity of technique.

In order to reduce variation in these factors to a minimum, twelve laboratories participated in the investigation of the relative efficiencies of four methods of homogenization. Each laboratory chose sputa which were likely to contain few tubercle bacilli, and used small inocula derived from an original volume not exceeding 2 ml. of sputum for each method. Thus, any lethal or growth-inhibiting action on the tubercle bacilli peculiar to the individual homogenizing agents should be made more evident. Culture medium was prepared at a central source. Löwenstein-Jensen medium in half-ounce screw-capped bottles sloped at a constant angle was used throughout the investigation.

Methods of Homogenization

3 per cent. sulphuric acid (acid method)...	(Method 1)
4 per cent. sodium hydroxide (alkali method)	(Method 2)
Jungmann's method	(Method 3)
Trisodium phosphate method	(Method 4)

The investigation was divided into six stages so that each laboratory should test the effect of each method of homogenization in combination with every other method. This plan permitted tests on each pair of methods of homogenization at two laboratories during each stage. Unfortunately, this ideal was not completely attained because, owing to administrative difficulties, a few laboratories were unable to complete the six stages.

The design of the experiment is shown in Table 1.

TABLE 1

Laboratory	1st Stage 52 specimens	2nd Stage 52 specimens	3rd Stage 52 specimens	4th Stage 52 specimens	5th Stage 52 specimens	6th Stage 52 specimens
	Methods	Methods	Methods	Methods	Methods	Methods
A	2 + 4	3 + 4	3 + 1	1 + 2	3 + 2	1 + 4
B	3 + 4	3 + 1	4 + 2	1 + 4	2 + 3	2 + 1
C	3 + 1	1 + 2	3 + 2	1 + 4	2 + 4	3 + 4
D	2 + 4	1 + 4	3 + 2	1 + 2	3 + 1	3 + 4
E	3 + 1	3 + 4	1 + 4	3 + 2	1 + 2	2 + 4
F	3 + 4	2 + 4	2 + 1	3 + 2	1 + 4	3 + 1
G	1 + 2	3 + 2	1 + 4	2 + 4	3 + 4	3 + 1
H	1 + 2	3 + 1	3 + 4	2 + 4	1 + 4	3 + 2
I	3 + 2	1 + 4	2 + 4	3 + 4	3 + 1	1 + 2
J	1 + 4	2 + 4	3 + 4	3 + 1	1 + 2	3 + 2
K	3 + 2	1 + 2	3 + 1	3 + 4	2 + 4	1 + 4
L	1 + 4	3 + 2	1 + 2	3 + 1	3 + 4	2 + 4

Selection of Specimens

To ensure a fair trial for each method it was decided that each stage should consist of 52 specimens per laboratory, or as near to this number as possible, and that of these approximately 75 per cent. should be microscopically negative and 25 per cent. should have been proved microscopically to be " scanty " positives.

Laboratories were asked to examine the 52 sputa in each stage over a period of 10 weeks at a rate of about 5 per week.

Although approximately 75 per cent. of each series of specimens were to be microscopically negative, it was considered desirable that as many as possible should come from cases of active tuberculosis and should be negative only because the bacillary content was too low for microscopical diagnosis by ordinary techniques. For this reason routine specimens sent in by general practitioners were excluded. Selected cases from out-patient tuberculosis clinics and hospitals were considered more likely to yield the type of specimen required, but in order to obtain sufficient material selection was extended to sanatoria, although it was realized that an entirely different type of specimen would thus be introduced into the investigation.

Collection of Sputa

The sources from which specimens were obtained were chest clinics, hospitals and sanatoria. The composition of the series is shown in Table 9.

Sputum was collected in sterile 1-oz. Universal Containers with caps fitted with non-inhibitory black rubber liners (red rubber may inhibit bacterial growth, McCartney, 1949), or in waxed containers (" Mono " containers).

In order that approximately 5 ml. of sputum should be available for examination its collection was permitted over any period up to 24 hours. Patients ready for discharge from sanatoria proved a useful source of material but they usually expectorated only small amounts of sputum which often had to be collected over 24 hours to provide sufficient for homogenization. In such cases if at least 2 ml. of the original sputum was available it was regarded as permissible to dilute it with sterile saline to provide the customary volume for the duplicate homogenizations. This procedure was not permitted if the sputum had dried in the container.

The maximum period permitted between the collection of a specimen and its inoculation on to culture medium was 48 hours. When possible this period was reduced to 24 hours. If storage up to 48 hours was necessary the specimen was kept at 4°C.

Specimens sent to the laboratory by post were accepted if they had not been more than one day in transit. No attempt was made to inhibit the growth of contaminating organisms by the addition of sulphonamides or antibiotics to the sputum.

Some laboratories experienced difficulty in obtaining a sufficient number of specimens of sputum in every stage. Use of sputum from the same patient was therefore permitted on up to three occasions in any stage. Sputum from the same patient could also be examined on a similar number of occasions at any subsequent stage.

Laboratory Methods

The following instructions were issued to all laboratories. They were mostly based on the addendum to the Medical Research Council's Report on Tuberculous Bacillaemia (Report, 1933), whose recommendations were re-affirmed in the report of the London Sector Pathologists (Report, 1945).

Avoiding false positive results

Care to be taken to avoid contamination with saprophytic acid-fast bacilli. The main sources of these saprophytes in the laboratory are dust, bark and rubber stoppers, rubber tubing, stale water, staining solutions and the zoogloeal growth inside cold water taps. These saprophytes may, of course, be already present in the material submitted for examination.

Preparation of smears

Nummular, purulent or caseous portions of sputum to be selected for the preparation of thin, evenly spread, smears.

Slides after spreading to be dried in air and fixed by heat.

Slides to be stained by the Ziehl-Neelsen method and counterstained with Malachite Green in a dilution of 1 ml. of 1 per cent. solution of the dye in 60 ml. of double distilled water, the counterstain being allowed to act for only 15 seconds in order to give the palest possible background. The use of tap water to be avoided throughout the staining procedure, all stains being prepared from double glass-distilled water. Stains to be kept in dust-proof bottles in small quantities. During staining sufficient heat to be applied to the slides to keep steam rising. The staining time to be not less than 3 minutes and, after homogenization with alkali, deposits to be stained for not less than 20 minutes. The use of staining jars to be prohibited.

Microscopy

All laboratories to use similar $\frac{1}{7}$th inch oil immersion fluorite objectives in conjunction with x 6 eyepieces in a binocular microscope.

Objectives to be wiped free of oil before examining each slide and immersion oil to be *dropped* on to the film in order to avoid transfer of acid-fast bacilli from the slide to the oil reservoir.

The same procedure to be followed also in examining the deposit obtained after homogenization.

A " scanty positive " sputum for the purpose of this investigation to be defined as one in which, on careful examination of 100 fields, 20 or less than 20 organisms or clumps of organisms morphologically resembling *Mycobacterium tuberculosis* were detected.

" Scanty positives " to be verified by a second opinion in all instances.

Homogenization

Portions of 2 ml. (±0·25 ml.) of sputum to be pipetted into each of two clean sterile 1-ounce universal containers fitted with non-inhibitory black rubber liners, using a wide-bore glass tube graduated by means of mercury and fitted with a 5 ml. teat. After being once used, the pipette to be discarded for destruction.

Since the first quantity of sputum drawn into the pipette may be more fluid, the first amount pipetted from the original container to be allotted alternately, in successive specimens, to the two methods of homogenization being compared.

All homogenizing solutions to be made with sterile glass-distilled water.

After each of the four methods of homogenization, the specimen to be centrifuged. Similar types of centrifuge to be used in all the laboratories so that the centrifugal force should be the same in each instance.

(1) *Homogenization by 3 per cent. sulphuric acid*—To the 2 ml. of sputum in the universal container add an equal volume of 3 per cent. v/v sulphuric acid and incubate at 37° C. Shake the container at 5-minute intervals by the clock until it is judged that homogenization is complete, but incubation not to exceed 30 minutes. After homogenization fill the container to the shoulder with sterile double distilled water (giving a dilution of approximately 1 in 7). Centrifuge the container at 3,000 r.p.m. for 30 minutes and decant the supernatant fluid. Use the deposit for inoculation without additional washing.

(2) *Homogenization by 4 per cent. sodium hydroxide*—To the 2 ml. of sputum in the universal container add an equal volume of 4 per cent. caustic soda and incubate at 37° C., shaking at 5-minute intervals by the clock. Immediately homogenization appears to be complete, fill the container to the shoulder with double distilled water to dilute the alkali. After centrifugation at 3,000 r.p.m. for 30 minutes decant the supernatant. Add one drop of sterile phenol red indicator to the deposit and adjust the pH to 7–7·2 by means of 8 per cent. hydrochloric acid.

(3) *Homogenization by Jungmann's method*

Solution A

Ferrous sulphate	20 g.
Concentrated sulphuric acid	20 ml.
Double glass-distilled water	180 ml.

Solution A keeps indefinitely.

Solution B

Hydrogen peroxide 20 vol.	5 ml.
Double glass-distilled water	95 ml.

Solution B is made up fresh on each occasion it is required for use.

To the 2 ml. of sputum in the universal container add 1·2 ml. of Solution A and 1·2 ml. of Solution B, using sterile pipettes for dispensing the solutions for each morning's or afternoon's work. Shake the container for 30 seconds and then allow to stand on the laboratory bench for 20 minutes, shaking at intervals. Centrifuge at 3,000 r.p.m. for 30 minutes and decant the supernatant fluid. Fill the container to the shoulder with sterile saline and centrifuge again. Decant the supernatant fluid and use the deposit for inoculation without neutralizing it.

(4) *Homogenization by trisodium phosphate*—The homogenizing agent is a 10 per cent. solution of the anhydrous salt or a 23 per cent. solution of the crystalline salt. To 2 ml. of the sputum in the universal container add 2 ml. of the homogenizing agent and incubate for 24 hours at 37° C. Shake the container at intervals throughout the day. After incubation fill the container with distilled water to the shoulder, agitate and centrifuge at 3,000 r.p.m. for 30 minutes. Decant the supernatant fluid, add one to three drops of sterile bromo-thymol blue (prepared as described in " Chemical Methods in Clinical Medicine ", Harrison, Third Edition, p. 553) and neutralize with 5 per cent. hydrochloric acid.

The supernatant fluid after centrifugation to be decanted into 2 per cent. lysol.

Microscopic examination of deposit

After homogenization by each method a small portion of the deposit remaining after decanting the supernatant fluid to be spread and dried upon new glass slides and stained.

It is already known that after homogenization sputa positive on direct film may be found negative on microscopy. This phenomenon may be due to the effect of the homogenizing agents on the acid-fastness of tubercle bacilli or, with scanty positives, merely to chance.

Sources from which it was anticipated that acid-fast artefacts might be produced were excluded with two exceptions: (1) Sputum containing blood not apparent to the naked eye (red blood cells are known to produce acid-fast artefacts), and (2) acid-fast material produced by the action of the homogenizing agent on the sputum.

Inoculation of medium

Whatever the method of homogenization employed, the deposit left for inoculation to be dispensed in approximately equal quantities into six tubes of Löwenstein-Jensen medium previously numbered in sequence. A pipette of not less than 3 millimetres internal diameter to be used for this purpose. The inoculum to be allowed to flow over the surface of the medium and then rubbed over with a wire loop. The tubes of medium to be incubated in an upright position.

Examination of cultures

Cultures to be examined weekly. Practical consideration limited the incubation period to six weeks, although a longer period occasionally produces additional positive cultures. In the first week, a period of five days' incubation to be regarded as one " week " but any shorter period to be carried forward to the following week, i.e., the first " week " might represent as much as eleven days. On some occasions there may be doubt on the day of examination whether a tube is positive. In such instances the tube not to be marked as positive on the record card until proved to be so by macroscopic examination in the following week.

RECORDS

With the assistance of Mr. L. E. Griffiths, Deputy Records Officer of the Nuffield Bureau of Health and Sickness Records, a record card was designed to show the required information. " Punch cards " were then prepared for subsequent analysis by Powers machines.

The following details were recorded on the card:—

1. Name of laboratory.
2. Name, age and sex of patient.
3. Reference number and date of specimen (a block of numbers was allocated to each laboratory to avoid duplicate numbering).
4. Source of specimen—from out-patient clinic, hospital or sanatorium.
5. Result of direct microscopic examination—positive or negative.
6. Methods of homogenization used.
7. Order of pipetting specimen in relation to methods of homogenization.
8. Medium batch number.
9. Culture result on each tube, showing week of becoming positive.
10. Presence of contamination.

From the beginning of the third stage, the results of microscopical examination after homogenization were recorded independently for each method.

RESULTS

Comparative results of culture after homogenization

A total of 3,167 sputa were examined. Of these, 2,472 were microscopically negative and 695 microscopically positive. As each sputum was examined by two methods, the numbers of examinations shown in Tables 2, 3 and 4 are double the number of specimens. Furthermore, the percentages shown in *Tables* 2, 3, 4, 7, 8 and 9 have been calculated directly on the total number of examinations. Since the numbers of examinations made by each laboratory varied, the percentages are thereby *weighted.* From the *success rates* of each individual laboratory, the mean success rate for each of the methods of homogenization has been determined and used for estimating its efficiency. These *straight averages* are statistically preferable.

TABLE 2

Comparative Results of Culture after Homogenization

Method	Negative by direct microscopy	Positive by direct microscopy	Total
3 PER CENT. SULPHURIC ACID			
Number of examinations ...	1,222	349	1,571
Number culture positive ...	267	287	554
Per cent. culture positive ...	21·8%	82·2%	35·3%
4 PER CENT. SODIUM HYDROXIDE			
Number of examinations ...	1,243	361	1,604
Number culture positive ...	356	311	667
Per cent. culture positive ...	28·6%	86·1%	41·6%
JUNGMANN			
Number of examinations ...	1,280	333	1,613
Number culture positive ...	250	268	518
Per cent. culture positive ...	19·5%	80·5%	32·1%
TRISODIUM PHOSPHATE			
Number of examinations ...	1,199	347	1,546
Number culture positive ...	202	264	466
Per cent. culture positive ...	16·8%	76·1%	30·1%

The differences between methods shown in Table 2 are best illustrated by the microscopically negative sub-group. In the microscopically positive group a high proportion of the sputa yielded positive cultures by both methods of homogenization. The effect of this in reducing apparent differences is also reflected in the combined series. In the same way, differences in the microscopically negative series have been rendered less obvious by the inclusion within this series of a considerable proportion of sputa which were culturally negative by the two methods of homogenization by which they were treated.

In Table 3 are given the results of culture of the microscopically negative series from which sputa negative by *both* methods of homogenization have been excluded, and in Table 4 sputa *positive* by *both* methods have in addition been excluded. In the latter table a comparison is made between the results given by each individual pair of methods.

TABLE 3

Culture results on microscopically negative sputa culturally positive by at least one method of homogenization

Method	Number of examinations	Number positive on culture	Percentage positive on culture
H_2SO_4 3 per cent.	340	267	78·6
NaOH 4 per cent.	394	356	90·3
Jungmann	335	250	74·8
Trisodium phosphate	311	202	65·2

Although a considerable number of specimens yielded different cultural results with the two methods of homogenization under comparison in each of the six combinations, the figures shown in Table 4 give an exaggerated impression of the differences between the four methods.

TABLE 4

Cultural results of microscopically negative sputa which were positive on culture by one method only

Methods compared	Number of examinations	Number of sputa positive by one method only	Number positive by method			
			1	2	3	4
1 and 2	446	62	17 (*27%*)	45 (*73%*)		
1 and 3	406	46	28 (*61%*)		18 (*39%*)	
1 and 4	357	43	33 (*77%*)			10 (*23%*)
2 and 3	421	45		34 (*76%*)	11 (*24%*)	
2 and 4	376	61		51 (*84%*)		10 (*16%*)
3 and 4	453	48			25 (*52%*)	23 (*48%*)

Microscopically positive specimens

The average success rates (see p. 204) for the four methods using all direct microscopically positive specimens are:—

	Per cent.
Method 1 (sulphuric acid)	82·8
Method 2 (sodium hydroxide)	83·8
Method 3 (Jungmann)	81·0
Method 4 (trisodium phosphate)	75·1

The differences between these figures are not statistically significant. The result shows that, working with a series of specimens from a variety of sources, about 80 per cent. which are scantily positive on microscopical examination will produce a positive culture by any of the four methods of homogenization employed. This is in accord with the findings reported by Cruickshank (1952).

Microscopically negative specimens

The success rates on culture of all direct microscopically negative specimens are as follows:—

	Per cent.		Per cent.
Method 1	21·5	Method 3	18·8
Method 2	28·7	Method 4	16·8

Here the differences are more striking, and some of them are well above the 1 per cent. level of significance. As regards differences between pairs of methods, Method 2 (sodium hydroxide) is significantly better than any of the other three methods (at the 1 per cent. level). Method 1 is not significantly better than Method 3, but is significantly better than Method 4 (at the 1 per cent. level). Method 3 is not significantly better than Method 4.

Microscopically direct negatives and positives combined

The success rates for the combined direct negative and direct positive specimens are as follows:—

	Per cent.		Per cent.
Method 1	34·8	Method 3	30·1
Method 2	40·6	Method 4	29·5

These results place the methods in the same order as those obtained with the direct microscopically negative specimens. The differences are still highly significant, but not as significant as in the direct negatives alone. Method 2 is still significantly better than any of the other three methods (at the 1 per cent. level). Method 1 is significantly better than Method 4 (at the 5 per cent. level). Method 3 is not significantly better than Method 4 (i.e. below the 5 per cent. level). In contrast with the findings on the direct negatives, the combined series shows that Method 1 is significantly better than Method 3 (at the 5 per cent. level).

Microscopically direct negatives which were culture-positive by one or both methods

This series of direct negatives, excluding all those which failed to produce positive cultures by either of the methods used, shows the same order of merit as in the other series; but as the population of true positives is unknown, the figures are not comparable with those shown above, and are unsuitable for statistical tests of significance. Success rates are as follows:—

	Per cent.		Per cent.
Method 1	77·8	Method 3	66·9
Method 2	89·3	Method 4	61·2

It is apparent that for all classes of specimens, the order of merit of the four methods of homogenization remains the same:—

First:	4 per cent. sodium hydroxide	Method 2
Second:	3 per cent. sulphuric acid	Method 1
Third:	Jungmann	Method 3
Fourth:	trisodium phosphate	Method 4

The relative differences are least—and statistically not significant—in the microscopically positive group.

The differences are greatest, and statistically highly significant, in the group of microscopically negative specimens.

A summary of the statistical analyses which have been carried out is given in the appendix.

Influence of the order of pipetting on the distribution of tubercle bacilli when dividing the original sputa

Allowance was made in the design of the investigation for certain factors which could be foreseen as likely to have a disturbing influence on the results of the experiment. One such factor was the order of pipetting the original sputum into two containers for each pair of homogenizing agents. That no disturbance of the results could be ascribed to this process can be seen from Table 5 in which all positive results are analysed according to the order of pipetting.

TABLE 5

Influence of the order of pipetting on the distribution of tubercle bacilli when dividing the original sputa

Methods under comparison	Order of pipetting	Numbers of sputa positive on culture					
		Sputa negative on initial microscopy		Sputa positive on initial microscopy		All sputa	
		1st quantity pipetted	2nd quantity pipetted	1st quantity pipetted	2nd quantity pipetted	1st quantity pipetted	2nd quantity pipetted
1 and 2	1 : 2	53 (1)	69 (2)	56 (1)	60 (2)	109 (1)	129 (2)
	2 : 1	57 (2)	45 (1)	43 (2)	40 (1)	100 (2)	85 (1)
1 and 3	1 : 3	48 (1)	48 (3)	38 (1)	38 (3)	86 (1)	86 (3)
	3 : 1	36 (3)	46 (1)	43 (3)	41 (1)	79 (3)	87 (1)
1 and 4	1 : 4	38 (1)	28 (4)	54 (1)	49 (4)	92 (1)	77 (4)
	4 : 1	23 (4)	36 (1)	53 (4)	59 (1)	76 (4)	95 (1)
2 and 3	2 : 3	62 (2)	52 (3)	57 (2)	46 (3)	119 (2)	98 (3)
	3 : 2	36 (3)	50 (2)	62 (3)	63 (2)	98 (3)	113 (2)
2 and 4	2 : 4	64 (2)	39 (4)	44 (2)	40 (4)	108 (2)	79 (4)
	4 : 2	38 (4)	54 (2)	36 (4)	43 (2)	74 (4)	97 (2)
3 and 4	3 : 4	40 (3)	45 (4)	38 (3)	36 (4)	78 (3)	81 (4)
	4 : 3	29 (4)	36 (3)	46 (4)	42 (3)	75 (4)	78 (3)
1st pipetting: Positives on culture ...		524	—	570	—	1,094	—
2nd pipetting: Positives on culture ...		—	548	—	557	—	1,105

The methods used are indicated in parentheses.

Distribution of tubercle bacilli in the homogenized material

The homogenized material from each specimen was equally distributed among the six culture tubes, so that each tube should have an equal chance of producing a positive culture provided that the viable tubercle bacilli were evenly distributed in the inoculum.

Nine laboratories numbered their culture tubes in the order in which they were inoculated with homogenized material. In Table 6 the totals are given of Tubes 1–6 which were positive in these nine laboratories by each of the four methods of homogenization.

It is apparent from Table 6 that the order of inoculation made no appreciable difference to the chance of any particular tube producing a positive culture. It may be assumed therefore that the viable tubercle bacilli were evenly distributed in the homogenized deposit.

TABLE 6

Distribution of tubercle bacilli in the homogenized material

Method	Positive cultures in tube number					
	1	2	3	4	5	6
Method 1	277	271	281	268	265	265
Method 2	290	281	294	289	295	283
Method 3	236	232	234	239	228	230
Method 4	204	179	193	185	181	193
All Methods ...	1,007	963	1,002	981	969	968

Time of recognition of positive cultures

With all methods, more cultures became positive during the third week than in any other. By the end of the third week, the percentages of positive specimens showing characteristic growth with the four methods were respectively:—

	Per cent.		*Per cent.*
Method 1	61·6	Method 3	58·4
Method 2	54·4	Method 4	44·1

By the end of the third week also, the following percentages of all positive tubes had already been recorded:—

	Per cent.		*Per cent.*
Method 1	59·5	Method 3	56·5
Method 2	52·6	Method 4	43·9

These figures, based on *all* positive specimens and tubes respectively, are loaded against the more efficient methods of homogenization. The reason for this is that these methods picked up a higher proportion than did the others of sputa likely to be recognized late on culture, because they contained particularly small numbers of tubercle bacilli. When sputa positive by only one of two methods are excluded from the analysis, the percentages of positive specimens by the end of the third week are all increased, though not equally, and now read as follows:—

	Per cent.		*Per cent.*
Method 1	67·4	Method 3	63·9
Method 2	61·0	Method 4	44·8

and the percentages of positive tubes:—

	Per cent.		*Per cent.*
Method 1	63·2	Method 3	58·7
Method 2	57·1	Method 4	45·1

The comparative times of recognition of positive specimens by the four methods are shown in Table 7, which takes into account only those specimens which were positive by two methods, and expresses the results as percentages of the totals positive by each method at the end of six weeks. This method of presentation gives a comparative picture, though even here a slight bias against Method 2 persists, as the number of specimens of this series positive by Method 2 was still greater than by any other method (Method 2, 488; Method 1, 447; Method 3, 432; Method 4, 393). The influence of this bias on the percentages recorded in Table 7 must, however, be small.

TABLE 7

Percentages of sputa positive by each method which had become positive within 2 weeks, 3 weeks, 4 weeks, etc., excluding specimens positive by one method of homogenization only

	Method 1	Method 2	Method 3	Method 4
	Per cent.	Per cent.	Per cent.	Per cent.
By week 2	18·6	13·1	14·1	7·4
By week 3	67·4	61·0	63·9	44·8
By week 4	89·3	86·8	86·6	77·4
By week 5	96·7	95·8	97·0	91·9
By week 6	100	100	100	100

Positive cultures were recognized earliest after homogenization by Method 1, and latest after Method 4. There was little to choose in this respect between Methods 2 and 3.

Advantage of employing two methods of homogenization

An advantage might be expected from the use of two methods of homogenization on each specimen. The sodium hydroxide method used in conjunction with each of the other three methods added the greatest number of positive cultural results (21·6–29·6 per cent.). When used with the sodium hydroxide method the three other methods of homogenization increased the number of positive cultures by less than 10 per cent. (6·6–8·8 per cent.). None of the three methods of homogenization was found significantly better than the others when used in combination with the sodium hydroxide method.

The improvement attributed to the use of a second method of homogenization may well be nothing more than what might be expected from the use of twice the amount of sputum and twice the number of culture tubes.

Examination of deposits after homogenization

Of 2,472 sputa which were microscopically negative by direct examination, 114 were reported microscopically positive on examination of the deposit after homogenization by one or both methods. As both methods gave positive results in 45 specimens, there was a total of 159 microscopically positive deposits. Culture confirmed the microscopic finding in 120 cases, or 75·5 per cent. Failure to confirm the microscopic finding by culture may be attributable to artefact, or may be a result of the lethal action of the homogenizing agent or of the effect on the tubercle bacilli of chemotherapy of the patient.

The proportion of these failures observed with the four methods were as follows:—

With 3 per cent. sulphuric acid treatment ...	4 out of 18
With 4 per cent. sodium hydroxide treatment	7 out of 51
With Jungmann's method	11 out of 34
With trisodium phosphate treatment ...	17 out of 56

Elimination of contamination

A basic property of an efficient homogenizing agent is the elimination from the specimen of secondary organisms which might obscure the result. The total number of tubes inoculated with sputa treated by each of the four methods, and the percentage of tubes which were contaminated, are shown in Table 8.

TABLE 8

Analysis of contamination rates by methods

Method	Number of tubes inoculated	Number of tubes contaminated	Percentage contaminated
Sulphuric acid (1)	9,423	461	4·9
Sodium hydroxide (2)	9,618	382	4·0
Jungmann's (3)	9,667	499	5·2
Trisodium phosphate (4)	9,276	708	7·6

The effect of source of specimen on culture findings

In routine laboratory work the varying results obtained in the culture of sputum for tubercle bacilli reflect the source of the specimen. In this investigation, 1,119 sputa were from sanatoria, 1,815 from chest clinics, and only 116 from hospitals (Table 9). Tubercle bacilli were grown from 38 per cent. of microscopically negative specimens from sanatorium patients, but only 22 per cent. of positive cultures were obtained from sputa sent in by chest clinics (*vide* Table 9).

TABLE 9

Composition of the series and distribution of positive cultures therein

Source	Micro-Positive and Negative Combined			Micro-Positive			Micro-Negative		
	Total Specimens	Culture Positive	Per cent. Culture Positive	Micro-Positive	Culture Positive	Per cent. Culture Positive	Micro-Negative	Culture Positive	Per cent. Culture Positive
Out-patient	1,815	678	37·4	394[1]	363	92·2	1,420[2]	315	22·2
Hospital	116	57	49·1	36	35	97·2	80	22	27·5
Sanatorium	1,119	548	49·0	242	213	88·0	877	335	38·2
Not known	117	41	35·0	27	23	85·2	90	18	20·0

Notes 1 *and* 2.—Because of an accident to the punch cards these figures, when added together, are 1 short of the total 1,815.

Temperature rises during centrifugation

The London Sector Pathologists (Report, 1945) noted that in a watery specimen 20–30 minutes' exposure to 4 per cent. NaOH at 37° C. might destroy all tubercle bacilli, whereas organisms in a thick muco-purulent specimen would resist such treatment for hours.

During centrifugation the temperature rises, and it was found that in six laboratories this rise had been considerable. The effect of this seems to be of such importance and wide application that a separate report on the effects of the temperature rise is being presented in a subsequent paper.

From specimens which had been treated with sodium hydroxide and one of the other methods, a total of 723 positive cultures was obtained. Of these, 667 followed sodium hydroxide homogenization. In the six laboratories which exposed their specimens to the risk of over-heating, 315 of 335 positive cultures were obtained by the sodium hydroxide method, whereas in the six laboratories where the heating was less, 352 of 388 positive cultures followed the sodium hydroxide method. It would seem, therefore, that heating during centrifugation following sodium hydroxide treatment did not have any serious lethal effect on tubercle bacilli under the conditions of this experiment.

DISCUSSION

In this investigation the use of specimens met with in routine diagnostic work was preferred to a more artificial quantitative technique such as that used by Corper and Cohn (1933), who employed diluted suspensions of tubercle bacilli. The selection of specimens was confined to sputa of patients known to be tuberculous or with definite clinical and/or radiological grounds for suspecting phthisis. Seventy-eight per cent. of the specimens examined were microscopically negative.

As has been found by recent workers (Cruickshank, 1952) about 20 per cent. of microscopically positive specimens failed to grow on culture. This is probably not due entirely to the lethal effect of the homogenizing agents. During the period of the investigation streptomycin and P.A.S. were being increasingly used for therapy, and though it was often impossible to collect accurate detailed information on this point, it is known that during and after treatment of the patient, sputa microscopically positive for acid-fast bacilli may fail to give positive cultures or to infect guinea-pigs. Among microscopically negative specimens the same factor must also operate.

The use of two methods of homogenization for each specimen was essential for the purpose of this investigation but the results provide no evidence that it would be worth while in routine work.

The design of the experiment does not permit any recommendation based on statistical evidence of the best number of culture tubes to be inoculated in routine work for each specimen.

The conclusions are valid only for the technique as described, including the use of Löwenstein-Jensen medium. It is possible that with variations in the technique or the use of different media different results might have been obtained, affecting the order of merit assigned to the various methods.

SUMMARY

An investigation has been made of homogenizing agents commonly used in the cultural examination of sputa for *Mycobacterium tuberculosis*. Four agents were compared: 3 per cent. sulphuric acid, 4 per cent. sodium hydroxide, Jungmann's iron-acid-peroxide, and trisodium phosphate. The investigation was undertaken by twelve public health laboratories working on a uniform plan.

Three thousand one hundred and sixty-seven sputa from patients attending chest clinics, or in sanatoria and hospitals, were each examined by two methods of homogenization. The specimens were selected as likely to contain few viable tubercle bacilli so as to reveal any differences between the four methods.

The success rates of cultures of microscopically positive specimens were all within the range 83·8 to 75·1 per cent., but the differences were not statistically significant.

Analysis of the findings on all microscopically negative specimens showed the following order of success: sodium hydroxide (28·7 per cent.), sulphuric acid (21·5 per cent.), Jungmann's method (18·8 per cent.), and trisodium phosphate (16·8 per cent.). Sodium hydroxide proved significantly better than any of the other methods to an extent that would not have occurred by chance once in a hundred times, i.e. at the 1 per cent. level. Sulphuric acid was not significantly better than Jungmann's method, but was better at the 1 per cent. level than trisodium phosphate. Jungmann's method was not significantly better than trisodium phosphate.

The totals of microscopically negative specimens included a large number of specimens for which there was no evidence that viable tubercle bacilli had been present. By excluding specimens that failed to yield growth of *Myco. tuberculosis* after either method of homogenization, there is left a population of microscopically negative sputa proven positive by cultural results following one or both methods of homogenization. When comparing the cultural success rates on this selected population the ranking of the four methods remains the same, with sodium hydroxide first (83·3 per cent. positive), sulphuric acid second (77·8 per cent.), Jungmann's method third (66·9 per cent.), and trisodium phosphate fourth (61·2 per cent.). Such index numbers cannot, however, be tested statistically for significance (see appendix).

The lowest rate of contamination of cultures, 4 per cent. of tubes inoculated, was found after treatment with sodium hydroxide. The highest rate, 7·6 per cent., followed the use of trisodium phosphate.

On all classes of specimens—microscopically positive, microscopically negative and the two combined—the order of success rates produced by the four methods was the same, i.e. 4 per cent. sodium hydroxide was found significantly superior to each of the other three methods; 3 per cent. sulphuric acid, Jungmann's method, and trisodium phosphate followed sodium hydroxide in that order. Three per cent. sulphuric acid proved significantly better than Jungmann's method and trisodium phosphate. There was little difference between the results with Jungmann's method and trisodium phosphate.

STATISTICAL APPENDIX

SUMMARY OF STATISTICAL REPORTS PREPARED BY MR. R. J. NICHOLSON, M.A., DEPARTMENT OF ECONOMICS, UNIVERSITY COLLEGE, HULL

The statistical problem has been to decide whether differences between methods of homogenization as shown by their different average-efficiency measures are significant, compared with the random fluctuation in the data, and allowing for systematic variations between the 12 participating laboratories and also for the differences dependent on the source from which each laboratory selected its specimens.

Method Efficiency

Two procedures for estimating method efficiency have been proposed:—

Procedure A—This measures success numerically by relating the total number of culture-positive specimens to the known population from which the specimens were obtained. This form of analysis has been applied to specimens positive on direct microscopy and separately to specimens negative on direct microscopy and finally to the population of both types of specimen combined. Procedure A is wholly satisfactory (in a statistical sense) and the results which follow its use reliable.

Procedure B—When it became clear that specimens positive by direct microscopy yielded only statistically insignificant differences between methods of homogenization, it became necessary to analyse findings on the microscopic negative specimens which had proved culturally positive after one or both methods of homogenization. In such a series, negative cultural findings could fairly be counted as failures against the method of homogenization concerned. Evidence was available (see Tables 5 & 6) that technically the distribution of infective material was even between any pair of methods of homogenization and also as regards each of the 12 culture tubes inoculated from them.

Analysis using Procedure B has also been carried out. The final success rates using Procedure B, unlike those based on Procedure A, are *not* absolute measurements and are indeed little more than indices for ranking the methods of homogenization in their correct order of merit. It is illegitimate to apply strict significance tests to qualitative statistical information of this type.

The following propositions have been established algebraically:—

(1) Application of Procedure B does not upset the order of merit between the methods.

(2) Application of Procedure B does tend to reduce the true range of variation.

Technique

The statistical technique adopted was that known as the Analysis of Variance (Fisher, 1947). The variance is the statistician's measure of variations within a set of data, and in its simplest form is defined as $\sum_{r=1}^{N} \frac{(x_r - \bar{x})^2}{N}$, where x_r represents the $_r$th observation in the data, $\bar{x}$ represents the arithmetic mean of all the observations, and N represents the total number of observations. In the present investigation, it was necessary to analyse the total variations measured by $\sum_{r=1}^{N} \frac{(x_r - x)^2}{N}$ into:—

(*a*) those explained by systematic differences between methods,
(*b*) those explained by systematic differences between laboratories,
(*c*) the remaining variations which may be attributed to purely random causes.

1. *Success rates using all microscopically negative specimens*

Success rates of the type described above were computed for all 12 laboratories for each of the four methods. An "average success rate" for each method was obtained by taking the unweighted arithmetical average of the 12 separate rates. The unweighted arithmetical average was used because the experiment

was designed in such a way as to give all participating laboratories an equal chance of contributing to the final result. It would not have been in conformity with this principle to have biased the results in favour of any particular laboratory by weighting the separate success rates by the actual number of specimens tested.

The average success rates of the four methods of homogenization were as follows:—

Method 2	Method 1	Method 3	Method 4
28·7 per cent.	21·5 per cent.	18·8 per cent.	16·8 per cent.

The results of the variance analysis were:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	968·4	322·8	20·8
Laboratories	11	4,533·1	412·1	26·6
Error	33	510·6	15·5	—
Total	47	6,012·1	—	—

The significance of the variance ratio was found by computing Fisher's parameter Z, defined as $\frac{1}{2} \log_e$ (variance ratio). Tables giving the values of Z at various levels of significance have been prepared by Professor Fisher and others.

The conclusions to be drawn from the analysis are these:—

(a) *Difference between methods*

The variance ratio 20·8 was found to be significant at the 0·1 per cent. level This is an extremely high level and it may reasonably be concluded that the differences shown by the average success rates indicate significant differences in method efficiencies.

The mean square attributed to " error " (with 33 degrees of freedom) may be used as a basis for testing the significance of differences between the means of pairs of methods. The results are as follows:—

Method 2. Significantly better than any of the other three (at the 1 per cent. level).

Method 1. Not significantly better than Method 3, but significantly better than Method 4 (at the 1 per cent. level).

Method 3. Not significantly better than Method 4.

(b) *Differences between laboratories*

The variance ratio 26·6 was found to be significant above the 1 per cent. level. This may be taken to show that the differences between the laboratories were also highly significant. The explanation lies in the variation between laboratories of the type of specimen employed.

2. *Success rates using all direct positives*

The average success rates were as follows:—

Method 2	Method 1	Method 3	Method 4
83·8 per cent.	82·8 per cent.	81·0 per cent.	75·1 per cent.

Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Services—Aug., 1952. *Vol. II.*

ERRATA

Page 204

The table showing the results of variance analysis (microscopically negative specimens) should be replaced by the following table:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	968·4	322·8	21·6
Laboratories	11	4,533·1	412·1	27·6
Error	33	490·6	14·9	—
Total	47	5,992·1	—	—

Paragraph (*a*), *2nd line:* "ratio 20·8" should read "ratio 21·6".

Paragraph (*b*), *2nd line:* "ratio 26·6" should read "ratio 27·6".

Page 205

The table showing the results of variance analysis (microscopically positive specimens) should be replaced by the following table:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	539·6	179·9	1·73
Laboratories	11	4,507·9	409·8	3·93
Error	33	3,439·9	104·2	—
Total	47	8,487·4	—	—

The table showing the results of variance analysis (microscopically negative and microscopically positive specimens combined) should be replaced by the following table:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	947·0	315·7	15·1
Laboratories	11	6,761·5	614·7	29·3
Error	33	689·8	20·9	—
Total	47	8,398·3	—	—

(16840) 9/52 D.L.

The results of the analysis of variance were:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	539·6	179·9	2·05
Laboratories	11	5,059·1	459·9	5·25
Error	33	2,888·7	87·5	—
Total	47	8,487·4	—	—

Three points emerge. First, the order of merit between the methods is the same as before, but here the differences are *not* significant. Secondly, these success rates are higher than those obtained from the population of direct negatives; and, thirdly, they are more closely bunched together.

This may be taken to show that, when direct positive specimens are used, there is nothing to choose between the methods of homogenization; whereas when direct negative specimens are used, the better methods do, in fact, show their superiority.

The differences between the laboratories, though less than before, were still significant.

3. *Success rates using direct negatives and direct positives combined*

For Laboratory C Method 1 the success rates for the two sets of data combined would be: $\frac{25+19}{108+27}=\frac{44}{135}=32{\cdot}6$ per cent.

The average success rates were as follows:—

Method 2	Method 1	Method 3	Method 4
40·6 per cent.	34·8 per cent.	30·1 per cent.	29·5 per cent.

The results of the variance analysis were:—

Source of Variations	Degrees of Freedom	Sums of Squares	Mean Square	Variance Ratio
Methods	3	955·7	318·6	13·6
Laboratories	11	6,672·5	606·6	25·8
Error	33	774·7	23·5	—
Total	47	8,402·9	—	—

Again there are the same differences as before, still highly significant though not so significant as with direct negatives alone.

The differences between pairs of methods were slightly changed:—

Method 2 significantly better than any of the other three (at the 1 per cent. level).

Method 1 significantly better than Method 3 or Method 4 (at the 5 per cent. level).

Method 3 not significantly better than Method 4 (i.e. below the 5 per cent. level).

Mr. Nicholson's final analysis, together with the tables and calculations on which it has been based, have been deposited for future reference by any interested person with the Royal Society of Medicine.

The Working Party are greatly indebted to Mr. R. J. Nicholson, of the Department of Economics, University College, Hull, for making numerous statistical analyses of the results at various stages of the investigation.

In the planning of the experiment, invaluable statistical advice was given by Professor Bradford Hill, Dr. J. O. Irwin and Dr. P. Armitage of the Medical Research Council's statistical unit.

Technical advice on many points was given by Dr. J. E. McCartney.

The Nuffield Bureau of Health and Sickness Records gave great assistance in designing the record card and preparing the punch cards. We are particularly grateful to Mr. L. E. Griffiths and Mrs. W. F. McKay for willing co-operation in making the mechanical analyses.

Many chest physicians, medical officers of hospitals and sanatoria, and medical officers of health have assisted by arranging for the provision of suitable specimens, and for supplying detailed information.

Thanks are due to the staff of the media room of the Oxford Public Health Laboratory, who were responsible for all the media used in the investigation, and also to the medical, technical and secretarial staffs of the participating laboratories.

References

Baker, F. J. (1951) *J. med. Lab. Technol.*, **9,** 160.
Beattie, M. (1949) *J. Lab. clin. Med.*, **34,** 733.
Byham, B. B. (1950) *Amer. J. clin. Path.*, **20,** 678.
Corper, H. J. and Cohn, M. L. (1933) *Amer. J. Hyg.*, **18,** 1.
Corper, H. J. and Stoner, R. E. (1946) *J. Lab. clin. Med.*, **31,** 1364.
Corper, H. J. and Uyei, N. (1927) *Amer. Rev. Tuberc.*, **16,** 299.
Cruickshank, D. B. (1952) T. H. Sellors and J. L. Livingstone, " Modern Practice in Tuberculosis ", p. 53. Butterworth & Co., Ltd., London.
Fisher, R. A. (1947) " The Design of Experiments ". Oliver & Boyd, Edinburgh.
Gifford, D., McKinley, F. and Hunter, C. A. (1951) *Amer. J. publ. Hlth.*, **41,** 164.
Griffith, A. S. (1914) *Brit. med. J.*, i, 1171.
Harrison, G. A. (1947) " Chemical Methods in Clinical Medicine ". Third Edition, p. 553. J. and A. Churchill, Ltd., London.
Jungmann, K. (1938) *Klin. Wschr.*, **17,** 238.
Löwenstein, E. (1924) *Wien. klin. Wschr.*, **37,** 231.
Lurie, M. B. (1923) *Amer. Rev. Tuberc.*, **7,** 332.
McCartney, J. E. (1949) Pers. comm.
Madsen, S. T. (1950) *Nord. Med.*, **44,** 1870.
Mullahy, M. E. (1950) *Amer. J. clin. Path.*, **20,** 672.
Nassau, E. (1942) *Tubercle*, **23,** 179.
Petroff, S. A. (1915) *J. exp. Med.*, **21,** 38.
Rasmussen, K. N. (1951) " Tuberkelbaciller i Expectorat ". Ejnar Munksgaard, Copenhagen.
Report (1933) " Tuberculous Bacillaemia: a Review ". *Spec. Rep. Ser. med. Res. Counc.*, No. 182, p. 98.
Report (1945) " The demonstration of tubercle bacilli in infected material ". *This Bulletin*, 1945, **4,** 202.
Spendlove, G. A., Cummings, M. M. and Patnode, R. A. (1949) *Amer. Rev. Tuberc.*, **60,** 628.
Starkey, H. and Aubert, E. (1950) *Canad. J. publ. Hlth.*, **41,** 27.
Sweany, H. C. and Evanoff, M. (1928) *Amer. Rev. Tuberc.*, **18,** 661.
van Vranken, M. (1947) *Ibid.*, **55,** 374.
Weed, L. A., Smith, C. R. and Cummings, M. M. (1951) *Amer. J. clin. Path.*, **21,** 673.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.
Tel.: REGENT 8411. Extn. 91.

II.—LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The July Bulletin was issued on 30th July.

Note.
Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(16424) Wt. 8221—7283 3,300 9/52 D.L.

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

SEPTEMBER 1952 Vol. 11

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, London, W.1.

THE GROWTH OF ADOLESCENTS

by

E. Janet Boothby, S.R.N.,

Mavis A. Guy, B.A.,

and

T. A. Lloyd Davies, M.D., F.R.C.P.,

from

Industrial Health Unit, Boots Pure Drug Co. Ltd., Nottingham,
and School Health Service, Nottingham.

Periodic medical examinations of large numbers of persons imply first, validated standards by which to judge individuals and, secondly, screening methods to eliminate healthy persons so that others may receive prolonged attention. These facts are often ignored by those who advocate periodic medical examinations as a means of preserving health. The extension of the duties of Appointed Factory Doctors to include annual examinations of young persons under 18 years of age employed in factories, warehouses and docks, shipbuilding, building and engineering construction, etc. (Factories Act, 1948), has accentuated the lack of both means and standards. Similarly, medical inspection in County Colleges when established will be hindered without validated standards. The present paper is an attempt to provide standards of growth for young persons working in industry. If such are available, growth or lack of growth might be used as a screening procedure.

Literature

Observations on the physique and growth of boys aged 14-18 years are infrequent and, in Great Britain, mostly confined to highly selected groups (Friend and Bransby, 1947). Earlier workers were content to observe mean heights and weights for a given age (Roberts, 1878). Boas (1930) realised that the value of such observations was limited by the unjustified assumption that for different persons the rate of growth should be the same. For a group of New England boys he calculated the rate of growth at different ages according to initial stature. Some of Boas' results, particularly his observation that the age of maximum growth influences the growth curve, are probably explained by variation in the age of puberty. Boas seems to have observed this but only refers indirectly to it. Ellis (1946 and 1948) noted that boys between 11 and 16 years in the same age groups but different maturity groups showed differences in growth pattern which could be traced back as far as 6 years of age. Boys in the higher maturity groups were heavier and taller than boys of the same age but in a lower maturity group. Growth curves based solely on chronological age tend to hide differences dependent on sexual development. These variations must be borne in mind when using growth curves as a standard of judgment for individuals.

Adolescent girls seem to have been subject to even fewer observations than boys, probably because groups of girls are less frequent and such as do occur are a recent innovation. Also, determination of sexual maturity is inexact. Apart from the difficulty of accurate determination of the age of menarche, which has been emphasised by Wilson and Sutherland (1951), the physiological significance of the menarche is open to doubt. Montague (1946) has argued with great clarity that the menarche is not maturity which

can only be said to be reached when reproduction can be undertaken with safety. The delay in the rise of the incidence of first pregnancies until 19 years of age of girls married at or before the age of menarche in circumstances where birth control is almost certainly not used, gives strong support to evidence derived from observation of mammals. Wilson and Sutherland (1949) in a survey of girls from Oxford town and county found little evidence that the height and weight of girls age 17-18 years varied according to menarche age. Menstruating girls were bigger at ages 11-12 years and 15-16 years than non-menstruating girls. In the former observations, these authors differ from Shuttleworth (1937). Jokl (1946) agrees with Shuttleworth that menstruating girls are bigger than non-menstruating girls: also the former tended to be stronger but to have less endurance than the latter. The work of Cluver and Jokl (1945) seems to have established beyond doubt the deleterious effect of the menarche on endurance, even though strength is increased.

Present Investigation

Since 1920 all young persons age 14-16 years employed at Boots Pure Drug Co. Ltd., Nottingham, have been required to attend a Day Continuation School. From April, 1948, when the school leaving age was raised to 15 years and the Day Continuation School became a College, attendance has been required of young persons aged 15-18 years. No exemptions were or are granted. All young persons are subject to a medical examination before starting work, and subsequently to annual examinations at school or college during the term of 13 weeks in which their birthday falls. Young persons with birthdays in vacations were examined the previous term. After a young person left school or college, arrangements were made for annual measurements in the birth month until 19 or 20 years of age (depending on administrative convenience). A few persons were measured until 21 years of age. About 10 per cent. defaulted at the subsequent measurements. This means that with few exceptions all measurements, including the first, were within one to two months of a young person's birthday. Ages given in this paper refer to the age on the appropriate birthday. The present paper is a review of measurements taken since May, 1941 (when earlier records kept by L. P. Lockhart were destroyed by enemy action).

All measurements were taken with the patient wearing " shorts " for boys or knickers and standard thin dressing gowns (weight 0.25 kilos) for girls. Measurements were taken by state registered nurses. Not more than three nurses were taking measurements during any one period of time. All nurses were trained in a standard procedure and acquired a high degree of accuracy (for height less than 0.25 cms. error). Height was recorded with chin pressed against the neck. Sitting height was measured after the patient had been positioned by flexing the trunk (with head and arms between separated and flexed knees) so that the buttock slid against the angle made by the floor and vertical measure. The back was straightened against the measure and the chin pressed against the chest. Weight was recorded on two beam machines, checked for accuracy each month. All measurements were in the metric system. The factor $Ht/3\sqrt{}$ wt (linear equivalent) may conveniently be determined from a chart.

Skin thickness was measured below the right scapula by constant pressure calipers, and maximum epicondylar width by hard pressure of blunt calipers against the epicondyles of the left humerus.

Volume measurements were taken with the boys nude and girls wearing a regulation one piece (occasionally two piece) bathing dress. The subjects lay in a tank of warm water, immersed except for nose and mouth with

the neck supported by the hand of one of us. Water which overflowed as the subject was immersed was weighed. The subject was breathing quietly. Measurements were taken between the hours of 10 a.m. and 12 a.m.

The age of menarche was determined by nurses practised in pre-placement examinations. Using an arbitrary calendar suitable for each child (e.g. related to family birthdays, school terms, special events), we believe the menarche can be determined with reasonable accuracy, certainly within a particular school term or holiday.

RESULTS

Girls

Social Status. No difference in the distribution of height and weight between girls employed in offices and factories has been detected. Such a difference would not be expected as during and since the war both have been recruited from families and schools with similar social, educational, domestic and occupational backgrounds.

TABLE I

Mean Measurements of Girls according to Chronological Age

Number of girls, 2,032

Age (years) ...	14	15	16	17	18	19	20	21	All ages
Number of observations ...	723	1,458	1,715	1,306	909	478	288	132	7,009
Weight (kilos)...	45·8	49·8	52·1	53·4	54·3	54·7	54·7	54·3	—
S.D.	7·1	7·3	7·2	7·2	7·4	7·9	7·5	7·5	—
Height (cms.) ...	156·8	159·4	160·5	161·0	161·4	161·1	161·4	161·2	—
S.D.	6·3	6·0	5·9	5·8	5·9	6·1	6·1	5·8	—
Sitting Height (cms.)	81·5	83·4	84·4	85·1	85·5	85·3	85·4	85·4	—
S.D.	3·7	3·3	3·1	3·0	3·1	3·1	3·1	3·1	—
Linear equivalent (Ht/3$\sqrt{}$wt)	44·21	43·55	43·15	42·91	42·77	42·64	42·64	42·1	43·12
Number of girls 1,001.									
Number of observations ...	—	148	353	309	191	—	—	—	1,001
Skin thickness (cms.) ...	—	0·6	0·7	0·7	0·7	—	—	—	0·7
Number of girls 719.									
Number of observations ...	—	141	269	207	102	—	—	—	719
Epicondylar width (cms.)	—	5·9	5·9	5·9	5·9	—	—	—	5·9

Mean measurements. Table I presents the mean weight, height, skin thickness and epicondylar width measurements according to age. Girls show a greater variation in weight than in height, and show a tendency to a slightly disproportionate increase in weight over the period 19 to 21 years of age. The distribution of observations of weight and height for each age group is Gaussian with insignificant positive skewing.

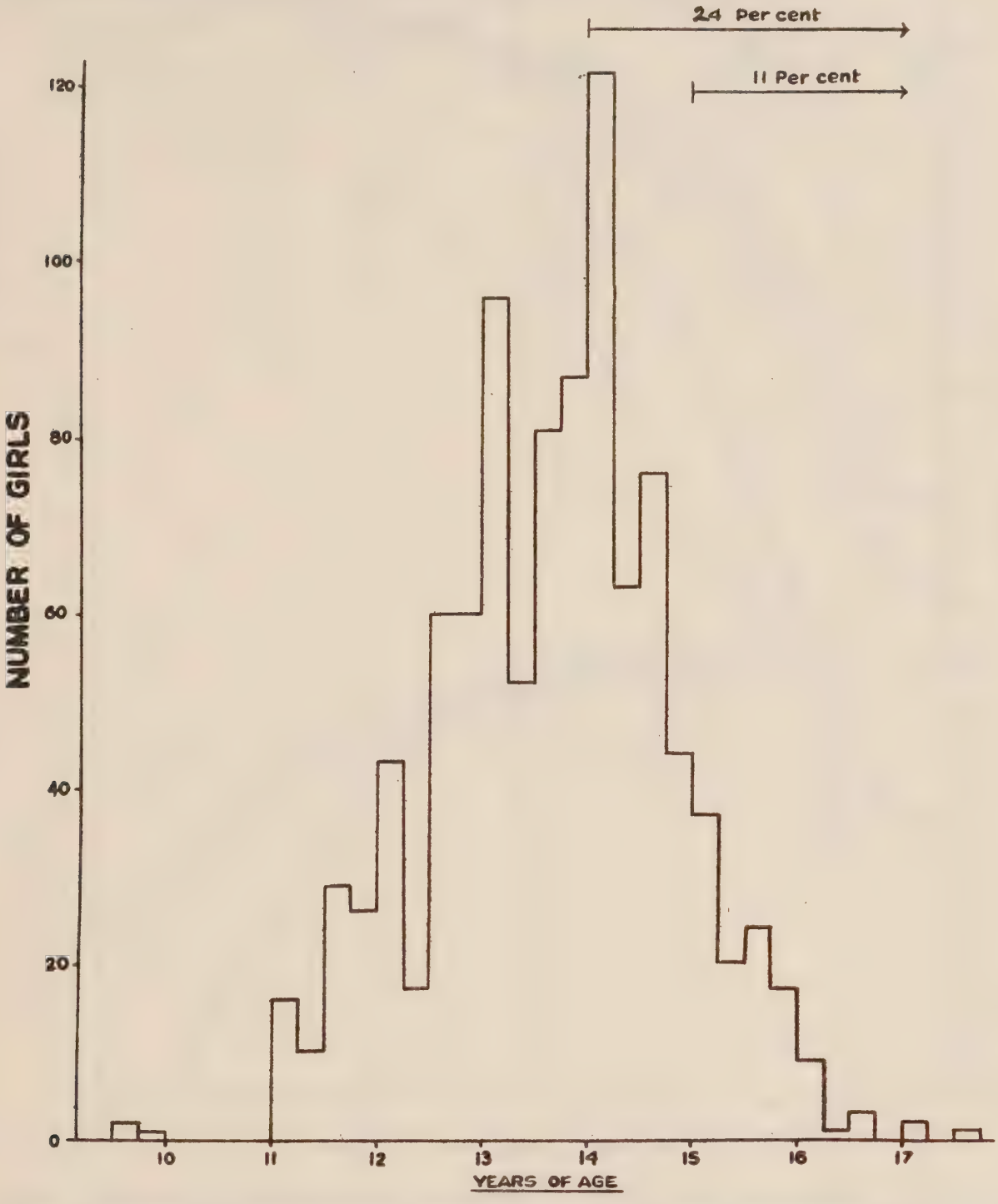


FIGURE I. Distribution of age of menstruation of one thousand girls.

Menarche. Figure I shows the age of menarche of one thousand girls. The reduction from 24 to 11 per cent. of girls non-menstruating when starting work by the rise in the school leaving age in 1948 from 14 to 15 years is not unimportant evidence that many girls at the younger age were too immature to enter gainful employment.

Effect of Menarche on Height and Weight

Figures II and III show that girls menstruating early are heavier, on average, at all ages than girls menstruating later. The latter, on average, catch up the former in height by 17-18 years of age.

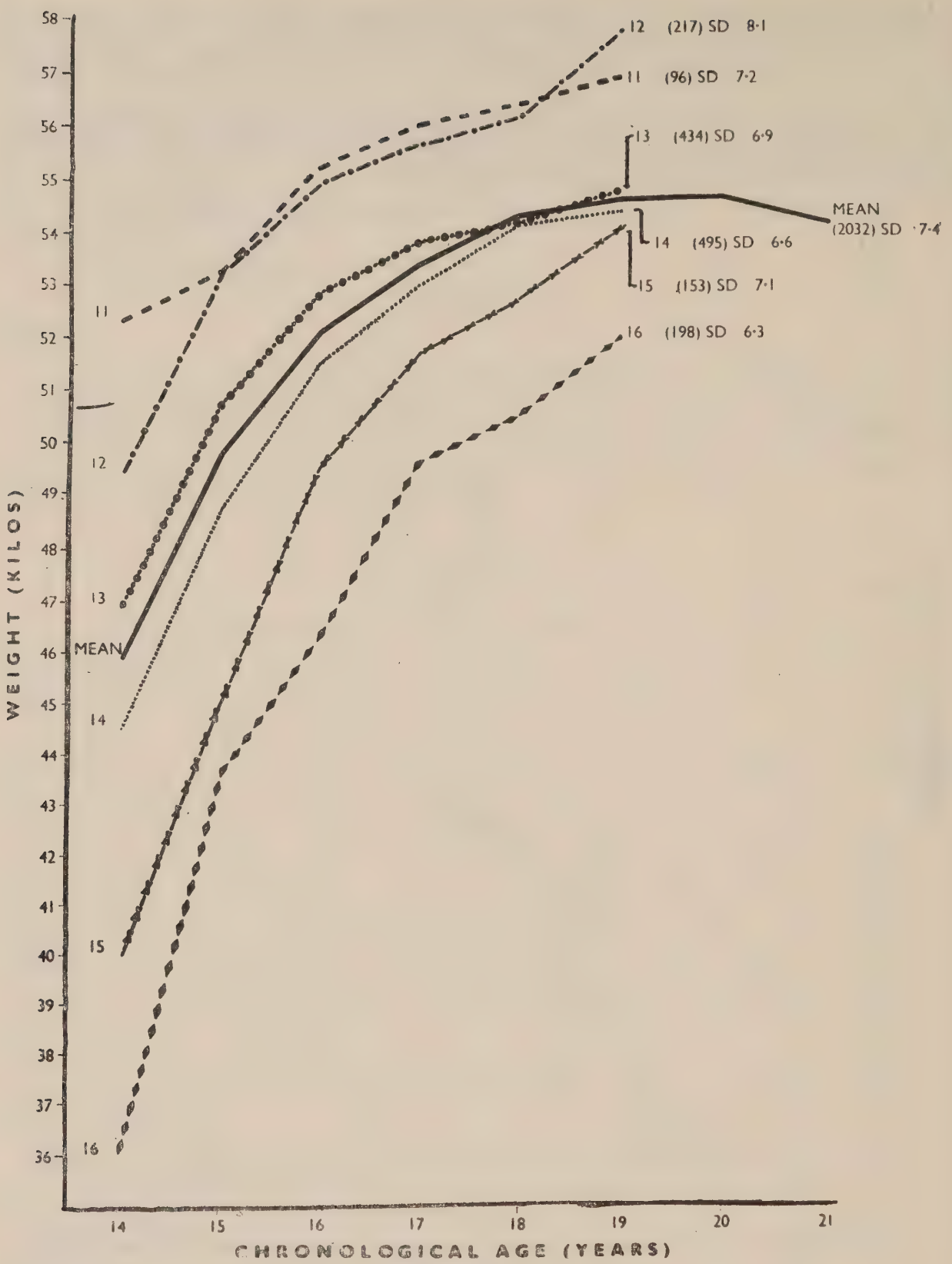


FIGURE II. Mean weights of 2,032 girls according to age, together with mean weights of girls with menarche at different ages. (Age of menarche, number of girls in each menarcheal age group and standard deviation indicated against each curve.)

Table II shows that at 18 years of age, when height and weight tables suggest maximum growth is reached, girls with early menarche tend to have a lower value for $Ht/3\sqrt{wt}$ (linear equivalent) and thicker skin than others. The mean epicondylar width was the same at all ages. These tendencies are confirmed by similar data in our possession for girls of all ages. Figure IV is a graph of skin thickness of girls of different menarche ages according to chronological age. The same tendency is demonstrated. Stress must be laid on the fact that relationship of low linear equivalents and early

menstruation is associative and not causal ; no firm opinion can be expressed but we are inclined to think both are the expression of another factor.

TABLE II

Girls age 18 years. Linear Equivalent, ($Ht/3\sqrt{wt}$) Skin Thickness and Epicondylar Width according to Age of Menarche

Age at Menstruation	Number of girls	Linear Equivalent	Skin Thickness (cms.)	Epicondylar Width (cms.)
10	3	39·67	1·2	—
11	12	41·54	0·8	5·9
12	24	41·87	0·8	6·1
13	64	42·30	0·7	5·9
14	61	42·51	0·7	6·0
15	14	43·64	0·6	6·0
16	2	43·5	0·5	6·2

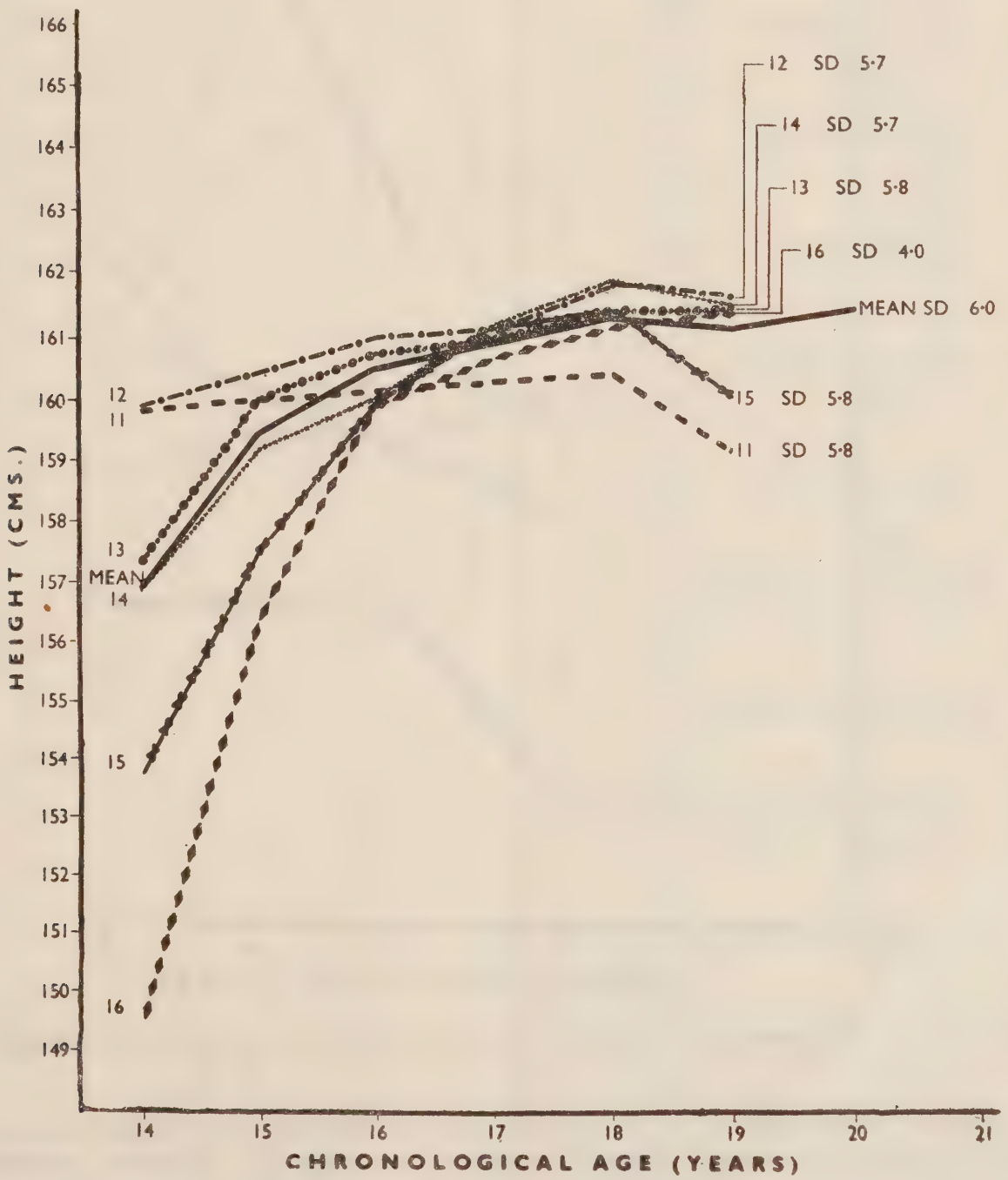


FIGURE III. Mean heights of 2,032 girls according to age, together with mean heights of girls with menarche at different ages. (Age of menarche and standard deviation indicated against each curve.)

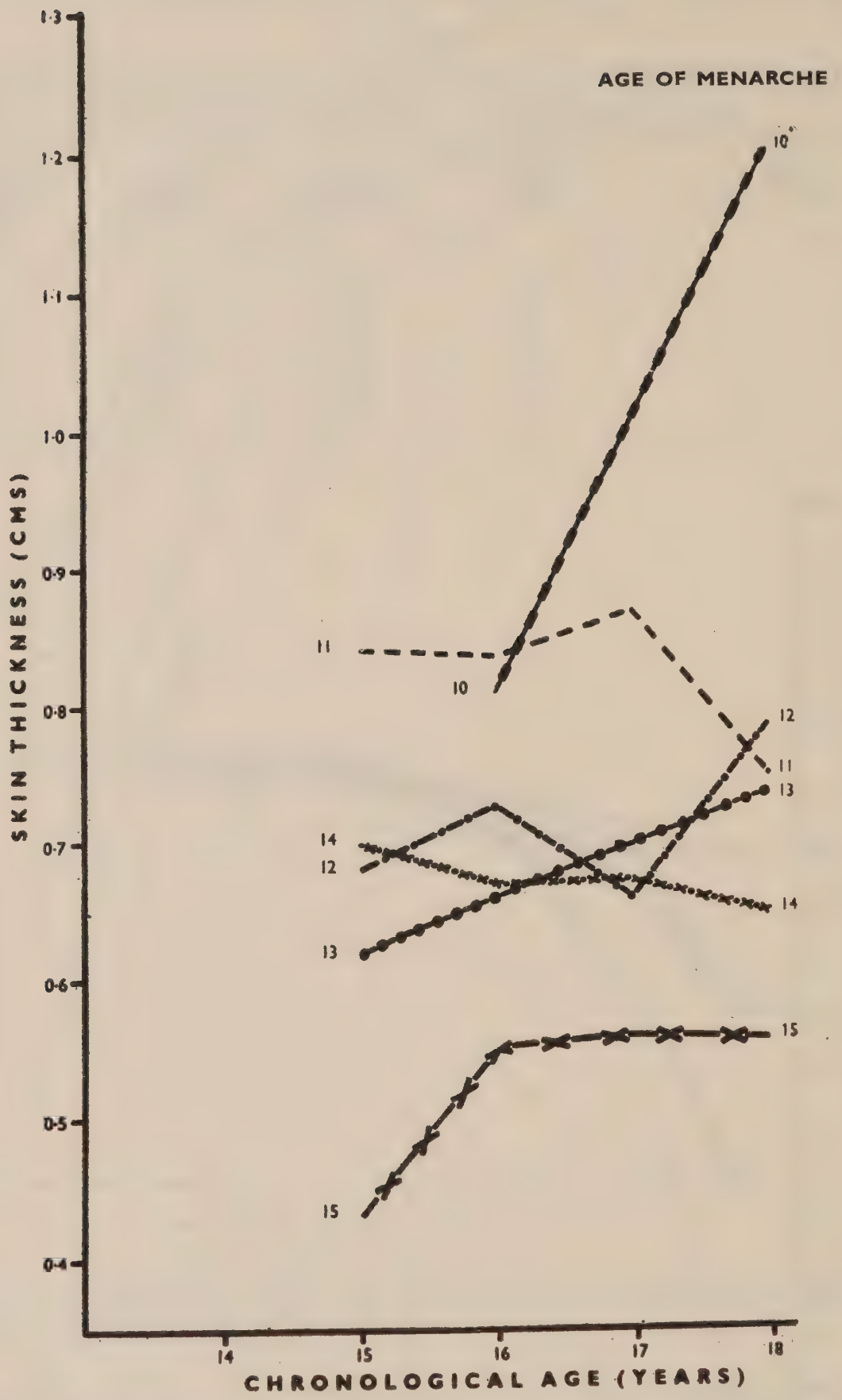


FIGURE IV. Skin thickness of girls according to chronological age and age of menarche.

Volume. Difficulties in the accurate determination of volume are such that too much reliance should not be placed on table III. It tends to confirm that the onset of menstruation is accompanied by an increase in volume bigger than accounted for by weight ; i.e. the body becomes less dense.

Table III

Volume of Girls according to Menstrual History

	Mean Age (years)	Range (years)	Number of girls	Ht. cms.	Wt. kilo.	Volume (litre)	Linear Equivalent Ht/ $3\sqrt{wt}$	Vol/ $3\sqrt{wt}$
Non-menstruating	13 2/12	11 7/12–14 11/12	63	148·8	40·6	36·6	43·26	10·6
Menstruating 1 year	14 1/12	12 1/12—15 1/12	32	155·1	48·1	44·9	42·61	12·3
Menstruating 2 years	14 1/12	12 6/12—14 11/12	13	156·3	49·6	46·1	42·47	12·5

Table IV

Increments in Height (cms.) of Girls

Year of Birth	Aged 14		Aged 15		Aged 16		Aged 17		Aged 18		Aged 19		Aged 20		Aged 21	
	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase
1925	—	—	4	1·6	10	2·28	—	—	—	—	—	—	—	—	12	·92
1926	1	1·3	16	2·01	37	1·92	7	·9	—	—	3	2·13	9	·3	16	·66
1927	—	—	87	2·05	69	1·67	11	·93	11	—·64	18	·71	31	·6	14	—·48
1928	—	—	49	1·89	47	1·72	16	·58	16	·26	31	·62	9	—·52	2	—·95
1929	7	1·93	57	1·93	54	1·37	40	·33	38	·89	24	·1	14	—·05	6	—·17
1930	2	·05	96	1·62	94	1·34	88	·44	62	·15	35	·29	15	·25	2	·45
1931	—	—	125	1·37	120	1·01	126	·51	130	·26	79	·04	37	·49	—	—
1932	1	1·3	136	1·21	133	1·39	146	·59	156	·31	117	—·01	—	—	—	—
1933	—	—	28	1·34	83	1·15	121	·56	129	·61	1	·5	—	—	—	—
1934	—	—	2	2·5	89	1·22	99	·83	—	—	—	—	—	—	—	—
1935	—	—	—	—	66	·93	1	·5	—	—	—	—	—	—	—	—

TABLE V

Increments in Weight (kilos) of Girls

Year of Birth	Aged 14		Aged 15		Aged 16		Aged 17		Aged 18		Aged 19		Aged 20		Aged 21	
	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase	Observations	Average Increase
1925	—	—	4	3·43	9	3·6	—	—	—	—	—	—	—	—	22	−1·04
1926	1	·45	19	2·31	46	2·7	10	1·01	—	—	3	·98	24	−·45	34	·01
1927	—	—	91	1·94	93	2·84	17	2·77	15	1·08	43	·39	69	−·5	33	·36
1928	2	2·49	54	1·34	60	2·45	27	1·33	32	·85	49	·11	28	·31	2	1·71
1929	8	1·57	58	1·85	64	1·95	56	1·27	62	·77	48	·47	17	·48	8	−1·61
1930	1	3·29	96	1·55	118	1·93	117	1·23	121	·34	51	·6	29	−·53	14	−·26
1931	1	3·4	129	1·35	156	1·52	176	1·32	163	·44	97	·56	68	·62	—	—
1932	1	·11	152	1·23	170	2·24	173	1·00	199	·76	146	·49	—	—	—	—
1933	—	—	41	1·32	91	2·52	144	1·25	165	·83	—	—	—	—	—	—
1934	—	—	—	—	111	2·69	125	1·6	—	—	—	—	—	—	—	—
1935	—	—	—	—	89	2·49	—	—	—	—	—	—	—	—	—	—

Yearly Growth. The annual increments in height and weight are shown in tables IV and V. Girls aged 16 years of age seemed susceptible to variations in growth and figure V shows their experience in different years. For them, 1947 was a particularly bad year. Variation in the growth of girls of this and other ages must be interpreted with caution. Before variation

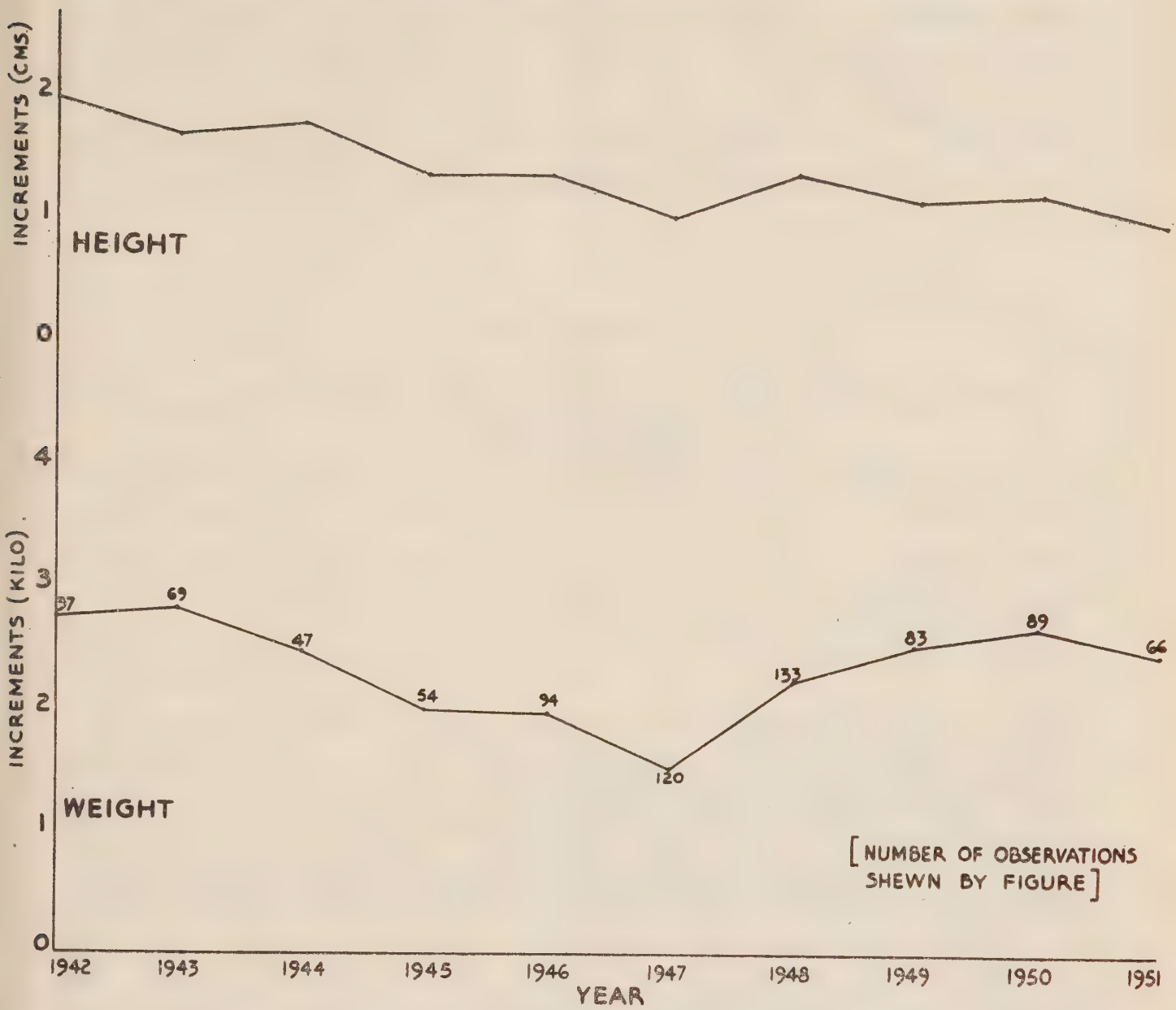


FIGURE V. Annual Increments in height and weight of girls age 16 years.

in the annual growth is ascribed to food supplies, many other causes must be eliminated. One of these may well be emotional stress (c.f. Widdowson 1951). All that can be said is that no cause is known.

Physical Types of Girls

The factor $Ht/3\sqrt{wt}$ will distinguish between ectomorphy on the one hand and endomorphy on the other. We suggest that this factor should be called the linear equivalent. Parnell (1951) has suggested the use of skin thickness to distinguish endomorphy and mesomorphy. The distribution of skin thickness and epicondylar width x linear equivalent for 1001 and 719 girls (age 15-18 years) respectively is shown in tables VI and VII. No significant variation in the distribution pattern or means occurred for age or age of menarche. The graph of skin thickness x linear equivalent shows that a break occurs in the curve at linear equivalent 39-40 and about

TABLE VI

Frequency of Linear Equivalent and Skin Thickness of 1,001 *Girls* (*Age* 15–18 *years*)

Linear equivalent	36	37	38	39	40	41	42	43	44	45	46	47
Number of girls...	4	7	12	27	91	142	233	214	164	76	25	5
Mean skin thickness (cms.) ...	1·5	1·4	1·4	1·1	0·8	0·8	0·8	0·7	0·6	0·5	0·5	0·6

TABLE VII

Frequency of Linear Equivalent and Epicondylar Width of 719 *Girls* (*Age* 15–18 *years*)

Linear equivalent	36	37	38	39	40	41	42	43	44	45	46	47
Number of girls...	2	4	8	21	56	100	179	150	112	66	16	4
Mean epicondylar width (cms.) ...	5·3	6·2	6·1	6·2	6·0	6·0	6·0	5·9	5·9	5·9	5·9	5·9

43-44. A linear equivalent 40-43 or 44 may be arbitrarily chosen as a normal range, above this is regarded as ectomorphic and below this range as endomorphic. Bullen and Hardy's (1946) table of incidence of somatotypes (Sheldon 1940) of North American women college students converted to metric measure, confirms this suggestion. In their table, endomorphic scores of 6 (and some scores of 5) fell below a linear equivalent of 40. Ectomorphic scores of 6 and 7 were above a linear equivalent of 44. Bullen and Hardy's girls with high mesomorphic scores (6 or more) fell within the range 40-43 chosen by us as normal range of linear equivalent. We have not been able to develop a simple measure of mesomorphy ; some of the girls in our sample with high muscular components (estimated on a clinical impression) had normal linear equivalents.

Similarly, a normal range for skin thickness may be chosen as from 1.1 to 0.6 cms. (or more selectively from 0.8 to 0.6 cms.).

The mean epicondylar width of 5.9 cms. (SD = 0.98) shows very little variation according to linear equivalent.

We believe a grid as shown in figure VI is a useful conceptual apparatus. At least the range of normality of physical types is defined. Whether further definitions such as ectomorph, normo-ectomorph etc. should be elaborated at this stage, we are doubtful. Indeed, without photographs the adoption of the Sheldonian terminology (except perhaps endomorphy and ectomorphy) is undesirable. Marked dysplasia between linear equivalent and skin thickness should draw attention to possible operation of other factors (e.g. factor concerned with age of menarche).

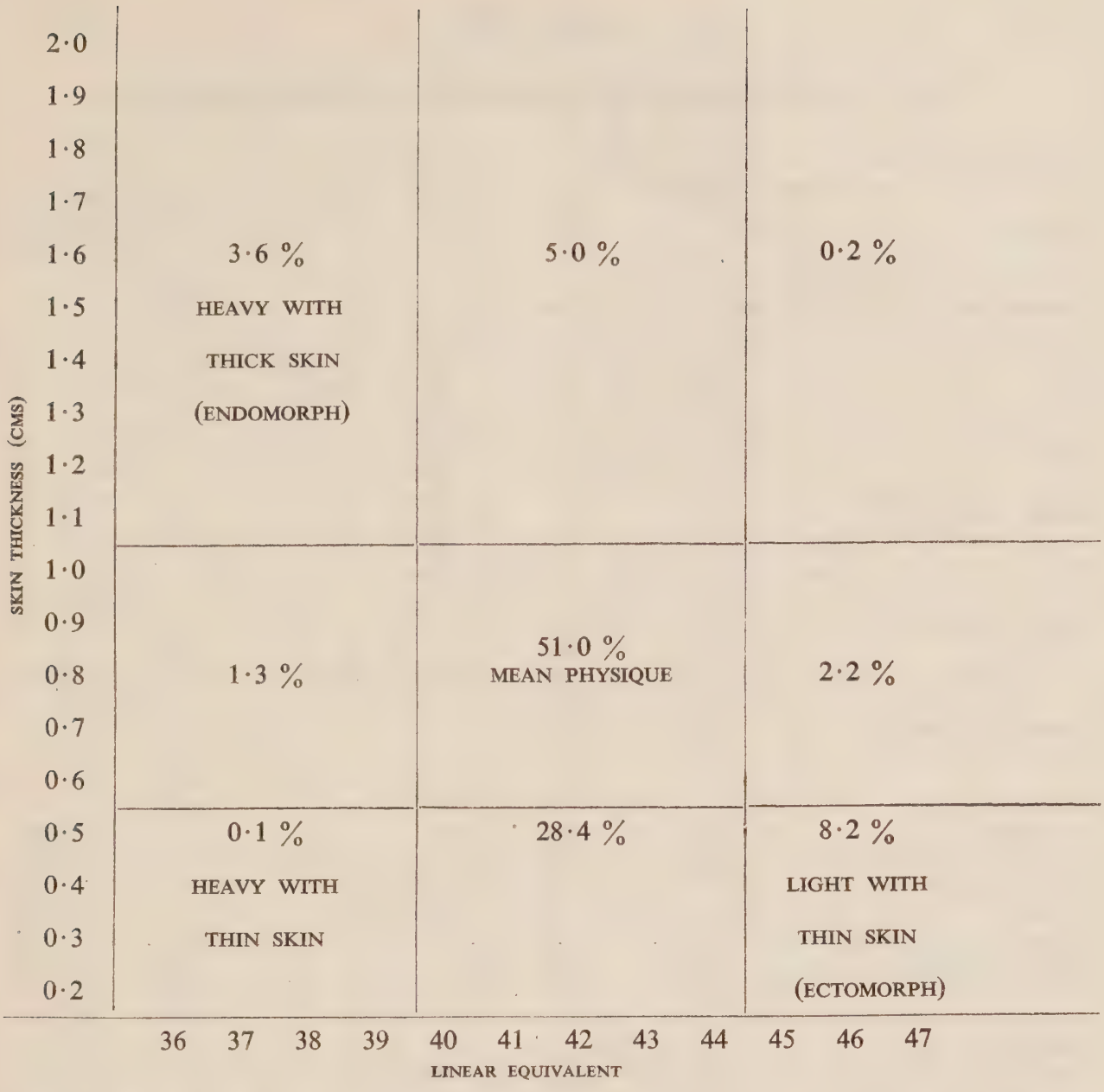


FIGURE VI. Grid showing frequency distribution of linear equivalent ($Ht/3\sqrt{wt}$) and skin thickness of 1,001 girls age 15–18 years. Percentages refer to distribution in each square.

Boys

Comparable data for boys are given in tables VIII, IX, X, XI, XII and XIII. Boys' heights and weights are not markedly greater than girls' at 14 and 15 years of age. Boys' heights tend to vary more than girls'. Because of the small number, the results do no more than suggest that arguments similar to those developed for girls are applicable.

Discussion

A clinician examining young persons needs standards by which to judge physical well-being or health. A useful measure is growth, whether judged by annual increment or by comparison of weight and height with "normal" for age. Difficulty may be experienced in judging whether apparent "underweight"—a term often used—is due to immaturity, smallness of stature, malnutrition or to disease. The tables presented above provide criteria for the assessment of girls and boys belonging to similar occupational, geographically distributed and social groups similar to those for which the tables were constructed. In so far as other groups do not differ markedly, they may be of wider value.

Table VIII

Mean Measurements of Boys according to Chronological Age

Number of boys, 537

Age (years) ...	14	15	16	17	18	19	20	21	All ages
Number of observations ...	240	444	482	308	170	36	68	25	1,773
Weight (kilos)...	45·3	51·1	56·1	58·8	61·1	64·5	66·0	67·4	—
S.D.	7·7	8·2	7·7	7·4	7·5	8·9	8·2	6·4	—
Height (cms.) ...	157·8	164·9	169·4	171·9	173·3	172·3	174·3	174·6	—
S.D.	7·8	8·0	7·3	6·9	6·8	6·8	7·0	5·4	—
Sitting Height (cms.) ...	80·0	83·5	86·3	88·3	89·5	91·1	89·9	90·8	—
S.D.	4·4	4·6	4·2	3·5	3·4	3·4	3·8	4·1	—
Linear equivalent (Ht./ $3\sqrt{}$wt.)...	44·56	44·49	44·29	44·27	44·18	42·96	43·33	43·22	44·28
Number of boys, 102. Number of observations ...	—	—	37	30	35	—	—	—	102
Skin thickness (cms.) ...	—	—	0·5	0·5	0·5	—	—	—	0·5
Number of boys, 52. Number of observations ...	—	—	22	11	19	—	—	—	52
Epicondylar width (cms.)	—	—	6·8	7·2	6·9	—	—	—	6·9

All measurements (except volume) forming the basis of this paper, were taken in the course of routine school medical examinations. This accounts for the many defects of procedure of which we are only too well aware. The isolation of three or four measures, height, weight, skin thickness (and epicondylar width), must lead to some artificiality. In clinical assessment many other factors are taken into account even if intuitively. Compared with standardised photographs, which in routine practice are seldom if ever possible, three or four measures must have disadvantages. In spite of these considerations, we suggest that for screening examinations, where reasonable speed is essential, the use of easily determined and reproducible measures, allows of the sorting of a large number of young persons. Also, tables, which may easily be constructed by anyone in charge of groups, enable nurses to select those young persons who are within normal range and to refer the others to a physician. Some such procedure both in this and other fields will become increasingly needed, if any progress is to be made in improving individual health. Tables do no more than allow of assessment in relation to mean values ; the essential process is good judgment.

TABLE IX

Volume of Boys according to Sexual Grade (Mean Measurements)

	Mean Age (years)	Range (years)	Number of boys	Ht. cms.	Wt. kilo.	Volume (litre)	Linear Equivalent Ht./ $3\sqrt{wt.}$	Vol./ $3\sqrt{wt.}$
Sexual Grade I	13 11/12	12 4/12—15 11/12	85	150·8	39·3	34·8	44·35	10·2
Sexual Grade II	14 6/12	12 5/12—17 17/12	43	158·4	46·1	39·6	44·16	11·05
Sexual Grade III...	15 8/12	13 7/12—18 11/12	67	167·4	55·8	47·8	43·9	12·5

TABLE X

Increments in Height (cms.) of Boys

Year of Birth	Aged 15		Aged 16		Aged 17		Aged 18		Aged 19		Aged 20		Aged 21	
	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase
1925	—	—	—	—	12	2·7	—	—	—	—	—	—	—	—
1926	—	—	23	5·06	13	2·71	—	—	—	—	1	−0·9	1	0·7
1927	15	6·31	24	6·57	11	3·52	—	—	—	—	1	0·7	—	—
1928	31	6·46	30	5·15	12	3·24	5	0·9	1	1·9	—	—	—	—
1929	25	6·76	34	5·0	22	2·22	17	0·94	1	0·6	—	—	3	0·6
1930	52	6·66	56	5·01	52	3·22	44	1·64	4	0·72	3	0·8	—	—
1931	17	5·99	20	5·63	24	2·96	20	1·67	6	0·32	1	3·5	—	—
1932	34	5·4	33	5·59	31	2·98	24	1·7	3	0·58	—	—	—	—
1933	9	7·1	37	3·92	45	2·77	35	1·94	—	—	—	—	—	—
1934	—	—	48	4·31	42	3·49	15	1·68	—	—	—	—	—	—
1935	—	—	26	4·15	6	3·11	—	—	—	—	—	—	—	—
1936	—	—	15	5·43	—	—	—	—	—	—	—	—	—	—

TABLE XI

Increments in Weight (kilos) of Boys

Year of Birth	Aged 15		Aged 16		Aged 17		Aged 18		Aged 19		Aged 20		Aged 21	
	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase	Obser-vations	Average Increase
1925	—	—	—	—	12	4·72	—	—	—	—	—	—	—	—
1926	—	—	24	5·08	14	2·09	—	—	—	—	—	—	—	—
1927	19	5·22	24	6·73	11	3·05	—	—	—	—	—	—	—	—
1928	31	5·26	30	5·17	12	3·18	7	3·14	2	1·6	1	−1·59	1	−2·80
1929	26	6·68	36	4·69	25	2·91	22	2·07	3	4·03	—	—	5	−0·75
1930	52	5·62	57	4·90	55	3·56	50	2·55	5	2·65	3	3·07	2	−2·42
1931	20	5·08	19	4·88	26	3·54	19	3·17	7	3·0	4	1·35	—	—
1932	34	4·85	35	5·96	31	3·38	27	2·67	6	4·27	—	—	—	—
1933	9	5·66	37	4·17	44	3·21	35	3·44	—	—	—	—	—	—
1934	—	—	48	4·79	47	3·63	16	2·91	—	—	—	—	—	—
1935	—	—	28	4·88	7	3·18	—	—	—	—	—	—	—	—
1936	—	—	15	5·23	—	—	—	—	—	—	—	—	—	—

Table XII

Frequency of Linear Equivalent and Skin Thickness of 102 *Boys*
(*Age* 15–18 *years*)

Linear equivalent ...	38	39	40	41	42	43	44	45	46	47	48
Number of boys ...	1	2	2	6	14	28	22	20	5	1	1
Mean skin thickness (cms.)	2·2	1·0	0·9	0·7	0·5	0·4	0·4	0·4	0·4	0·6	0·3

Table XIII

Frequency of Linear Equivalent and Epicondylar Width of 52 *Boys*
(*Age* 15–18 *years*)

Linear equivalent ...	38	39	40	41	42	43	44	45	46	47	48
Number of boys ...	1	1	0	3	5	16	12	9	3	1	1
Mean epicondylar width (cms.)...	7·3	8·2	—	7·1	6·8	7·0	6·8	6·8	6·6	6·4	6·8

We are indebted to Dr. A. A. E. Newth, O.B.E., Senior School Medical Officer and Mr. F. Stephenson, Director of Education, Nottingham, for much help ; to Miss K. Scraton and Miss F. Towers for many tedious calculations ; to Mrs. C. M. Ginnever for patient assistance ; to Miss M. P. Griffiths and the late Mr. C. W. Lawrence for providing some of the subjects for measurement of volume. Especially, we wish to thank Mr. F. W. Coe and Miss E. M. Wood of Boots College for unfailing courtesy and assistance with many tiresome requests.

References

Boas, F., 1930. Science. (U.S.) **72,** 44.
Bullen, Adelaide K. and Hardy, Harriet L. 1946, Amer. J. Phys. Anthropology, (N.S.), **4,** 37.
Cluver & Jokl, E. 1945, South African J. Med. Science, **10,** 105.
Ellis, R. W. B., 1946, Arch. Dis. Child, **21,** 17.
Ellis, R. W. B., 1948, Arch. Dis. Child, **23,** 181.
Friend, G. E. & Bransby, E. R., 1947, Lancet, **ii,** 677.
Jokl, E., 1946, Nature, **157,** 195.
Montagu, M. F. Ashley, 1946, Adolescent Sterility, Illinois.
Roberts, C., 1878, Manual of Anthropometry, p. 72, London.
Sheldon, W. H., 1940, The Varieties of Human Physique, New York.
Shuttleworth, F. R., 1937, Monograph of the Society for Research in Child Development, 2, No. 5. NRC Washington.
Wilson, D. C. & Sutherland, I. 1949, Brit. Med. J., **11,** 130.
Wilson, D. C. & Sutherland, I. 1951, Brit. Med. J., **26,** 198.

THE USE OF GENERAL PRACTICE RECORDS IN STUDYING MORBIDITY

By W. P. D. Logan, M.D., Ph.D., B.Sc., D.P.H., Chief Medical Statistician, General Register Office

Commenting on an article by Dr. John Fry entitled "A Year of General Practice: A Study in Morbidity", the British Medical Journal says that Dr. Fry has shown what can be done in a busy practice and deserves all praise, and expresses the hope that others will follow his example. Dr. Fry bases his paper on the records of his own practice in the year 1951 and states its objects as:—

(1) To present a picture of the work of a general practice as recorded by facts and figures collected over a period of one year.

(2) To examine the causes of ill health in the particular community under consideration.

(3) To stress the conditions which are most often encountered in general practice.

(4) To show that a so-called large practice can be managed easily and, it is hoped, efficiently.

(5) To indicate the large scope which exists for research in general practice.

Others who have likewise studied general practice records recently and published reports are Pemberton (1949) and McGregor (1950).

It may be of general interest that a pilot enquiry designed to discover how far records maintained in general practice might actually be used on a somewhat wider basis to provide useful information of this kind, but of more general applicability, was instituted by the General Register Office and the Ministry of Health at the beginning of 1951. It was known that enthusiastic practitioners who believe in the value of good records could keep them in a form suitable for statistical analysis. It was, however, realised that the maintenance of such records must normally mean some extra burden on the doctor, that it might be difficult to find a sufficient number of willing doctors with practices of different types for their amalgamated experience to be representative of general practice in England and Wales, and that there might be certain types of practice where it would prove impracticable to maintain records in a suitable form.

The pilot enquiry was planned, therefore, to cover a variety of practices, primarily with a view to seeing whether, for example, size or situation of practice were associated with any special difficulty in record keeping; to testing different methods of recording; and to finding out how much, if any, extra trouble would be involved.

Names of practitioners likely to be interested were supplied by Regional Medical Officers of the Ministry of Health; the aims and methods of the enquiry were discussed with several of them and they were asked to co-operate. The first practitioner began keeping records for the pilot enquiry on 1st January, 1951, and by 1st April, 1951, ten practitioners were doing so. One of these ceased to keep the records at the end of the year, but two further practitioners have joined the enquiry during 1952 including Dr. Fry, the writer of the article referred to above.

No attempt was made to cover a representative cross-section of general practice in England and Wales but nevertheless a fairly wide variety of types of practice is covered. One of the practices concerned is a partnership and three employ assistants, a feature which complicates uniform record keeping. In size they range from just under 2,000 to some 4,000 patients, with one practice (the partnership) dealing with 7,600 N.H.S. patients. Two of the smaller practices are entirely rural in character, one of them having a large influx of hop pickers in the summer months; one is in a highly industrial town; two in country towns of a mixed character; one is near a seaside resort; and the remaining five are in the Greater London area.

The form and method of recording have, to some extent, varied from practice to practice in order to meet the particular circumstances of each and to afford an opportunity of comparing the relative merits of the different methods. The forms of record used in most of the practices are, however, based on the official National Health Service record card (E.C. 7 or 8) either as it stands or with some modification, and entries are normally made in accordance with a simple code. In each practice details of every consultation, except those given to private patients, are recorded under five headings: Date of consultation; Place of consultation; Certificates issued; Referrals to hospitals, etc.; and Diagnosis (or other reason for consultation).

While the main objects of the present pilot enquiry relate to the keeping of records, problems of classification and tabulation are also being considered, and tabulation relating to a year's experience in eight of the practices should have been completed by the end of this year. The proposed tables are designed to show, for different diagnoses:—

(1) Illness in relation to the doctor's work, e.g., numbers of consultations, certificates issued, and referrals made for various reasons.

(2) Illness in relation to the patient, e.g., sex and age, and frequency of consultation.

The collection of comprehensive and reliable morbidity statistics is an ideal which, unlike mortality statistics, may never be completely achieved. In the Survey of Sickness the Ministry of Health and the General Register Office, with the help of the Social Survey organization, took the logical step of going to those who actually suffer from ill health—a sample of the general public—to ensure that every degree of ill health was covered in the enquiry. One of the main limitations was uncertainty about statements of diagnosis which were not made by doctors. The closest medical contact with the patient is that of his general practitioner and, while the Survey of Sickness showed that there is a vast amount of ill health which does not receive regular medical attention, it may be that, for practical purposes, his contact with his patients in their illnesses is close enough for his records to provide much of the data required for morbidity surveys. This is certainly likely to be true for many diseases for which the patient will almost invariably consult his doctor. Thus, if sufficient doctors can find time and enthusiasm to maintain records in a suitable form, these records will perhaps provide the best source of information for investigating the variations in incidence of disease, as well as a standard against which individual doctors can measure their own experience.

The present pilot enquiry has already confirmed that the problems to be overcome are many and important, but it would be a pity to rely entirely on individual enthusiasts presenting their personal experience piecemeal.

That is why the Ministry of Health and the General Register Office and its Medical Advisory Committee thought it well worth while making an attempt to co-ordinate these efforts in such a way that information of general applicability representing the sickness experience of a wider community could be made available.

References

Brit. Med. J. (1952) Annotation, **ii**, 276.
Fry, J. (1952) Brit. Med. J. **ii**, 249.
McGregor, R. M. (1950) Edinburgh Med. J. **lvii**, 433.
Pemberton, J. (1949) Brit. Med. J. **i**, 306.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, AUGUST, 1952

Issued from the General Register Office, Somerset House, W.C.2

	August 2nd	August 9th	August 16th	August 23rd	August 30th	Average weekly figures, August, 1951
Scarlet Fever	997	722	643	559	557	514
Whooping Cough	2,043	1,766	1,872	1,806	1,851	2,920
Diphtheria	19	19	17	23	22	26
Measles, excluding Rubella ...	7,317	7,012	6,099	5,256	3,649	3,545
Acute Pneumonia	228	239	247	207	220	237
Meningococcal Infection	20	18	35	29	28	27
Acute Poliomyelitis (Paralytic) ...	139	167	170	152	150	57
,, ,, (Non-paralytic)	91	83	88	69	65	60
Ophthalmia Neonatorum... ...	34	19	34	39	28	33
Puerperal Pyrexia and Puerperal Sepsis	227	242	242	244	216	212
Dysentery	93	119	125	119	90	167
Paratyphoid	130	63	40	58	44	44
Typhoid	5	4	4	13	4	8
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Taunton Laboratory : Change of Address

The laboratory has moved to new quarters, and the address is now: Public Health Laboratory, Musgrove Park Hospital, Taunton (Tel: Taunton 5753 and 3662).

LABORATORY METHODS IN THE DIAGNOSIS OF ALASTRIM

A. W. Downie, M.D., D.Sc., K. McCarthy, M.D., and A. Macdonald, M.D., The Department of Bacteriology, University of Liverpool.

Earlier reports (Downie 1947, MacCallum 1952) have shown that laboratory methods are invaluable in confirming a clinical diagnosis of smallpox. The outbreak of alastrim in Lancashire during the spring of this year provided an opportunity to assess the value of laboratory tests in this disease and also to make observations on the most suitable materials for investigation. Although alastrim and smallpox are epidemiologically distinct from one another, the two viruses appear to be serologically indistinguishable and it has been found that the techniques used in the diagnosis of smallpox are equally effective in alastrim. Specimens were received directly by this department from 154 patients, and in 87 the diagnosis of alastrim was confirmed by one or more tests ; in two clinical cases a single examination was negative. Most of the negative specimens came from cases of atypical chickenpox and, though many of these could probably have been excluded on clinical grounds, there were others in which laboratory methods alone could eliminate the diagnosis of alastrim.

Technical Methods

Smears were obtained by scraping the base of papules or vesicles and transferring the material to clean glass slides. They were fixed with alcohol in the laboratory and then stained by Gutstein's methyl violet method (Gutstein 1937). Egg culture was carried out in the usual manner, using as inocula material from extracted smears or crusts, or capillary tubes of vesicle or pustule fluid (Downie and Dumbell 1947). Eggs were examined after 2-3 days' incubation. Complement-fixation tests for alastrim antigen were carried out with a rabbit anti-vaccinial serum in the same way as in smallpox. The antigen was prepared by extracting smears, pustule fluid or crusts. Rabbit vaccinial antigen or variola crust antigen was used for control tests. The technique employed for these tests was based on that of Craigie and Wishart (1936) and has been previously described (Downie and Macdonald 1950). Complement-fixation tests for antibody in the patient's serum were carried out with a rabbit vaccinial antigen and/or an alastrim egg antigen.

Results

Table 1 shows the number of specimens examined by each method and the number of positive tests. The decision as to which methods of examination were to be used in any case was determined largely by the type and amount of material available and the stage of the patient's illness. Whenever possible, however, egg culture was used in addition to any other method.

It is apparent from the figures in Table 1 that the microscopic examination of stained smears collected as a routine by medical attendants is, by itself,

TABLE 1

Results of Laboratory Tests on 89 Clinical Cases of Alastrim

	Nature of Test			
	Microscopic examination of smears	Complement-fixation test for antigen	Egg culture	Complement-fixation test for antibody
Number of specimens ...	40	31	75	22
Specimens positive ...	23	26	73	18
Specimens negative ...	17	5	2	4

insufficient to confirm a clinical diagnosis of alastrim. The method is still employed because a positive smear may allow the laboratory to give an immediate presumptive positive report in a suspected case. Such a report, must, however, be supplemented by the result of egg culture or some other test. A negative microscopical examination is of no value, although it must be mentioned that when smears are properly taken from lesions which have not progressed beyond the vesicular stage they invariably show large numbers of virus particles. Almost all of the 17 negative smears in Table 1 contained numerous red blood cells and were thus unsuitable for microscopical examination. It is useless to examine smears prepared from lesions which have become pustular.

By using the complement-fixation test to detect alastrim antigen in material derived from the patient's skin lesions a report can usually be given within 24 hours of receipt of the specimen. Table 1 shows that in 5 of 31 clinical cases the tests for alastrim antigen were negative. The five negative specimens were smears on glass slides (Table 2) and in these the material was in-

TABLE 2

Results of Complement-fixation Tests for Antigen and of Egg Culture in relation to the type of material examined

Nature of test	Result	Type of material examined			
		Smears on slides	Vesicle fluid in capillary tube	Pustular fluid and crusts	Totals
Complement-fixation test for antigen	+	5	5	16	26
	—	5	0	0	5
Egg culture	+	58	3	12	73
	—	1	0	1	2

sufficient as the smears had previously been extracted for egg culture. Although the number of complement-fixation tests for specific antigen is small, Table 2 shows that material from the vesicular to the crusting stage of the focal lesions may be successfully employed for these tests. Extracts prepared from vesicle fluid or from crusts in cases of alastrim are always positive provided sufficient material is sent for examination.

The complement-fixation test for antibody in the patient's serum can only usefully be employed after the 8th or 9th day of illness. This test is thus of no value for diagnosis in the early stages. Moreover it is impossible to distinguish a positive test which is the result of recent vaccination from one

caused by infection with alastrim. None of the 18 positive samples of serum listed in Table 1 was obtained from a vaccinated person, and the four negative results were obtained from patients on the 2nd, 5th, 6th and 9th days of illness. The complement-fixation test for antibody is of particular value in making a retrospective diagnosis in unvaccinated persons.

Tables 1 and 2 show that egg culture is an extremely sensitive test in that virus was grown from 73 of 75 cases of alastrim. Both failures can be explained because inadequate material was sent for examination; one of these cases had 2 and the other not more than 6-8 skin lesions. It can be seen from Table 2 that most of the positive results were obtained from material on slides, but vesicular or pustular fluid and crusts are equally satisfactory. (It was found, however, that when saline extracts of such specimens were sent through the post the virus died out rapidly. Several such samples were sent by Dr. Parker of the Public Health Laboratory, Manchester, in order to have his serological findings confirmed by cultural methods and these tests are not included in Table 1.)

As in smallpox, egg culture is undoubtedly the most sensitive method for laboratory diagnosis of alastrim and its only disadvantage is that 2-3 days are required before a result is available.

As already mentioned, alastrim and smallpox viruses are serologically indistinguishable and it is thus impossible to decide by laboratory tests whether a patient is suffering from smallpox or from alastrim. In examining eggs inoculated with material from cases of alastrim we have noticed that the size of lesions produced is more variable than with variola. Often the lesions are indistinguishable from those due to the virus of smallpox, but sometimes they are much smaller and then resemble the lesion due to herpes simplex virus. Histologically, however, the alastrim lesions on the chorio-allantois are identical with those caused by smallpox virus and quite different from those caused by herpes simplex virus in this tissue. It may be mentioned here that one case of generalized vaccinia was investigated during the outbreak; vaccinia lesions in eggs are, of course, readily distinguishable from those caused by alastrim or smallpox virus.

Comment

The results of the present investigation confirm the value of laboratory methods in the diagnosis of alastrim. Of the 67 patients with negative laboratory findings, 65 were finally considered on clinical grounds not to be suffering from the disease, while all of the 87 patients who gave positive laboratory tests were accepted as clinical cases. It is clear that the clinician should not consider the diagnosis of alastrim to have been excluded when he receives a negative report on a stained smear. Both the complement-fixation test for alastrim antigen and the egg culture method are extremely reliable provided that there is sufficient material for examination. The test for specific antigen can provide a result within a day of taking the specimen while egg culture, although slower, is probably the most conclusive. Complement-fixation tests for antibody in the patient's serum are only valuable when the patient has been ill for some 10 days, and it is thus chiefly used in atypical missed cases when the skin lesions have healed As in smallpox, the reliability of all the diagnostic procedures used in alastrim depends primarily on the submission of adequate and appropriate material for examination.

Summary

Laboratory methods are of great value in the diagnosis of alastrim, if satisfactory specimens are submitted. A negative report on a stained smear from a lesion cannot exclude alastrim. The complement-fixation test for

antigen, and egg culture, are very reliable. The former can give a result within a day; the latter is slower but probably more conclusive. Complement-fixation tests for antibody in the patient's serum are of value after the 10th day of illness.

We are grateful to Dr. J. Innes, Medical Officer of Health of Rochdale C.B., Dr. D. C. Liddle, Physician Superintendent of Monsall Hospital, Manchester, and Dr. R. W. Farquhar, Medical Officer of Health of Heywood M.B., for sending us the specimens and for information about their patients.

References

Craigie, J. and Wishart, F. O. (1936) *Canad. publ. Hlth. J.*, **27**, 371.
Downie, A. W. (1947) *Publ. Hlth., Lond.*, **60**, 82.
Downie, A. W. and Dumbell, K. R. (1947) *J. Path. Bact.*, **59**, 189.
Downie, A. W. and Macdonald, A. (1950) *Ibid.*, **62**, 389.
Gutstein, M. (1937) *Ibid.*, **45**, 313.
MacCallum, F. O. (1952) *J. R. sanit. Inst.*, **72**, 112.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health,
Savile Row (Room 207),
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The August Bulletin was issued on 29th August.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(16809) Wt. 8221—7283 3300 9/52 D.L. H.E. 4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

OCTOBER **1952** **Vol. 11**

CONTENTS

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, London, W.1.

SICKNESS AND INCAPACITY

Eileen M. Brooke, M.Sc. Statistician, General Register Office

For some years the Survey of Sickness, conducted by the Central Office of Information for the General Register Office has collected a variety of information about illnesses in samples of the adult population. Data for the years 1947–1950 are now presented, relating to the numbers of illnesses recorded and the amount of incapacity they have caused.

Each person chosen for interview was asked about their sickness experience in the two months preceding the interview, so that each contributed two person-months experience to the total. During 1947 and 1948 the average number of monthly experiences relating to persons aged 16–64 was about 2,270 for men and 2,760 for women ; during 1949 and 1950, the size of the monthly sample having been increased, the average was 2,943 for men and 3,500 for women.

Definitions

(i) *Incapacity :* inability of employed persons to go to work, or for housewives and unoccupied persons, confinement to the house.

(ii) *Sickness rate :* the number of persons of specified sex and age who reported some illness or injury during the month of experience, per 100 persons interviewed.

(iii) *Incapacity rate :* the number of days incapacity in the month per 100 persons interviewed.

(iv) *Consultation rate :* the number of medical consultations in the month per 100 persons interviewed. For the most part they comprise family doctor consultations in the home or the surgery.

Table 1 shows the monthly sickness, incapacity and consultation rates for men and women of 16–64, that is, of what may be taken to be the working ages.

TABLE 1

Sickness, Incapacity and Consultation Rates per 100 *Persons aged* 16–64 *interviewed,* 1947–1950

		Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sept.	Oct.	Nov.	Dec.
1947													
Sickness ...	M	65	65	60	55	57	54	53	53	58	62	62	62
	F	74	71	69	67	67	64	62	62	68	71	72	72
Incapacity ...	M	162	150	127	101	89	67	62	63	75	97	97	96
	F	154	140	120	78	79	54	51	55	56	66	92	95
Consultations	M	48	45	40	37	33	28	32	31	32	36	37	35
	F	45	38	41	39	38	35	35	34	33	35	48	41
1948													
Sickness ...	M	60	60	58	55	55	56	55	53	57	61	64	65
	F	68	69	69	66	67	66	67	66	69	72	73	75
Incapacity ...	M	106	119	104	83	88	85	74	77	85	102	109	92
	F	90	93	72	65	64	59	74	62	67	100	102	113
Consultations	M	36	39	41	33	34	35	32	31	29	35	42	38
	F	39	41	42	36	35	39	39	35	41	48	44	45

		Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sept.	Oct.	Nov.	Dec.
1949													
Sickness ...	M	64	66	66	61	60	57	56	55	55	60	64	63
	F	74	76	76	73	71	69	68	68	68	73	73	72
Incapacity ...	M	113	125	120	80	88	84	80	83	79	88	101	98
	F	108	136	135	88	63	72	76	65	70	96	104	93
Consultations	M	41	47	41	32	38	36	36	33	35	40	36	37
	F	51	54	51	47	41	41	46	42	42	45	45	42
1950													
Sickness ...	M	66	66	62	62	59	57	59	57	60	64	62	66
	F	75	73	73	71	71	69	68	68	71	74	72	74
Incapacity ...	M	109	115	110	89	88	88	80	73	80	92	84	113
	F	119	119	105	83	72	70	61	56	66	76	91	120
Consultations	M	37	46	47	41	37	36	33	32	40	38	35	40
	F	51	54	51	42	46	48	40	39	41	48	49	43

Invariably more women reported having had an illness than men, though the range in each sex was approximately the same, male rates varying between 53 and 66 and female between 62 and 76. Likewise women had more consultations than men (except for three months in 1947). On the other hand incapacity rates of men were frequently greater month by month than those of women during 1947, but female rates were higher than male during the winters of 1948–49 and 1949–50. Incapacity rates were also the most variable, ranging between 62 and 162 for men and 51 and 154 for women. (See Fig. I.)

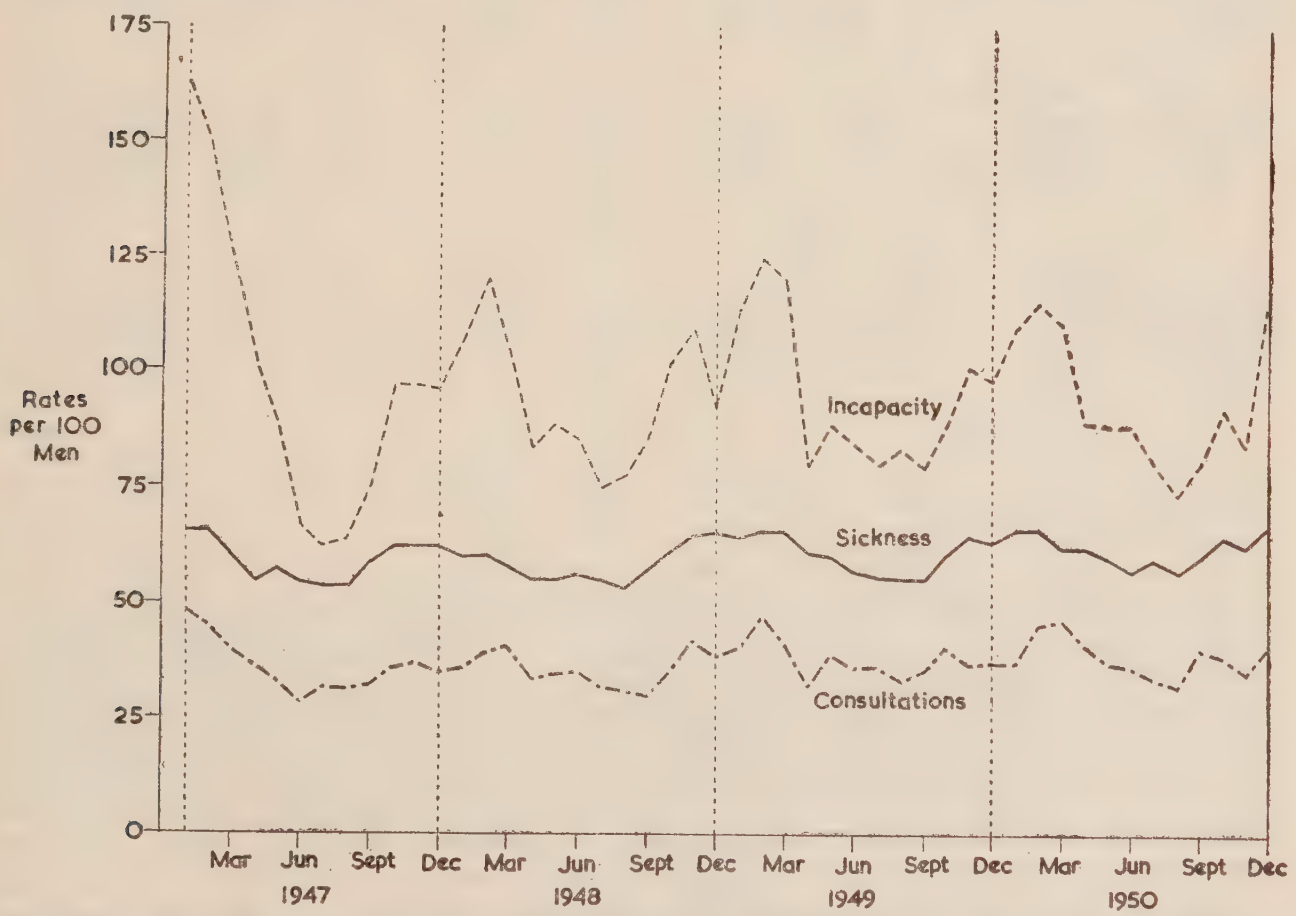


FIG. I.—Monthly Sickness, Incapacity and Consultation Rates per 100 men aged 16–64.

Occupational Comparisons

Table 2 shows details of sickness rates, medical consultations and days of incapacity for men and women aged 16 and over in different occupational groups. In calculating the number of days incapacity allowance has been

made for the variation in the number of days in the month and also for the fact that in stating the number of days of incapacity, Sundays would often be included but not usually holidays, the last column is roughly comparable with the usual index of working days lost per hundred possible days in the year(1).

TABLE 2

Sickness, Medical Consultation and Incapacity experience of persons aged 16 and over, according to Occupation. 1947–50

Occupational Group	Average Monthly Sickness per 100 Persons		Medical Consultations per person in year		Days of Incapacity			
					Per Person per Year of 12 standard months		Per 100 possible days	
	1947–48	1949–50	1947–48	1949–50	1947–48	1949–50	1947–48	1949–50
Males								
Manufacturing	60	62	4·1	4·3	11	10	3·4	3·1
Transport and Public Services	56	57	4·5	4·0	12	9	3·6	2·8
Mining and Quarrying...	66	67	6·3	7·2	21	24	6·5	7·4
Building and Roadmaking	57	59	3·6	3·8	10	9	3·1	2·9
Agriculture and Fishing	54	57	2·6	2·7	8	7	2·5	2·2
Distributive	58	59	3·4	3·8	8	8	2·4	2·4
Clerical	56	59	3·8	3·9	8	9	2·5	2·7
Professional and Managerial	57	60	3·5	3·6	7	8	2·0	2·3
Other employment ...	58	61	3·7	4·4	10	10	3·2	3·2
Total occupied males	58	60	3·9	4·1	10	10	3·1	3·0
Females								
Manufacturing	64	68	4·3	5·0	14	13	4·2	4·1
Distributive	64	64	3·7	4·3	11	8	3·2	2·6
Clerical	58	63	3·9	4·1	10	10	3·0	2·9
Professional and Managerial	59	62	4·3	4·3	9	9	2·8	2·8
Housewives	74	77	5·1	6·0	11	12	3·3	3·6
Total occupied females	70	73	4·8	5·6	11	11	3·3	3·4

As regards various types of employment for men, mining and quarrying stands out as having the highest rates in each two-yearly period. During 1947–48 and 1949–50, the average yearly loss per person due to sickness or injury was 3 to $3\frac{1}{2}$ weeks, as compared with not more than $1\frac{1}{2}$ weeks for other occupations. Of other groups, men in agriculture and fishing had low sickness rates and the lowest consultation rates. Professional and managerial personnel lost the smallest proportion of possible working time. Among females, housewives had the highest sickness and consultation rates whereas those engaged in manufacturing had the most incapacity. Women engaged in manufacturing, distribution, clerical, and professional and managerial occupations, the only four groups distinguished other than housewives, had higher rates of sickness, consultations, and incapacity than men in the nominally corresponding occupational groups.

(1) Registrar General's Quarterly Return, No. 402. H.M.S.O. 1949.

Duration of Sickness

The proportionate distribution of illnesses according to the number of days incapacity caused is shown in Table 3, together with the total number of illnesses and injuries and the total days of incapacity. There is in each quarter a peak value at 7 days. For example the distribution of illnesses causing from 4 to 10 days of incapacity in the first quarter of 1947 was :

Number of days ...	4	5	6	7	8	9	10
Proportion of illnesses per 1,000	4·8	2·7	2·5	13·2	1·6	1·0	4·3

TABLE 3

Proportionate Distribution of Illnesses and Injuries according to the days of incapacity caused. Persons aged 16 and over

	Proportion of Illnesses and Injuries causing Incapacity of the specified number of days during the quarter									Total Illnesses and Injuries	Total Days In-capacity
	0	1–3	4–6	7	8–10	11	18–	25–	Total		
1947—											
1st Quarter	918	20	10	13	7	12	7	13	1,000	25,721	24,531
2nd Quarter	953	14	6	6	2	7	4	8	1,000	21,395	11,398
3rd Quarter	961	15	4	5	2	6	2	5	1,000	20,945	8,237
4th Quarter	940	22	8	9	4	7	4	6	1,000	24,146	13,248
1948—											
1st Quarter	939	19	9	9	3	8	5	8	1,000	22,814	14,387
2nd Quarter	956	14	5	7	3	6	2	7	1,000	22,046	10,284
3rd Quarter	955	16	6	6	2	6	3	6	1,000	22,374	10,081
4th Quarter	935	23	9	8	5	9	4	7	1,000	26,521	15,859
1949—											
1st Quarter	913	25	14	12	8	13	6	9	1,000	38,393	31,919
2nd Quarter	940	17	9	8	6	9	4	7	1,000	34,526	21,023
3rd Quarter	945	16	7	7	5	10	3	7	1,000	30,995	18,248
4th Quarter	915	25	14	11	9	13	4	9	1,000	29,406	23,891
1950—											
1st Quarter	917	21	11	14	9	15	5	8	1,000	29,603	24,898
2nd Quarter	942	15	7	8	6	11	4	7	1,000	33,446	20,830
3rd Quarter	947	17	7	7	4	8	3	7	1,000	32,640	17,170
4th Quarter	918	29	12	11	6	11	5	8	1,000	34,667	25,024

If the experience of corresponding quarters be combined, so as to minimise the effects of abnormal conditions in any one year, the average duration of incapacity for illnesses causing some incapacity was 10.3, 10.6, 10.2 and 9.2 days in the four quarters respectively. The percentage of the total incapacity attributable to illnesses of short duration was as follows:—

	Duration of 1–3 *days*	4–6 *days*	7 *days*
First quarters	5·3	6·6	10·3
Second quarters	5·1	6·0	9·0
Third quarters	6·2	5·7	9·0
Fourth quarters	7·2	7·7	10·2

Between a fifth and a quarter of total days of incapacity was due therefore to illnesses of up to a week's duration.

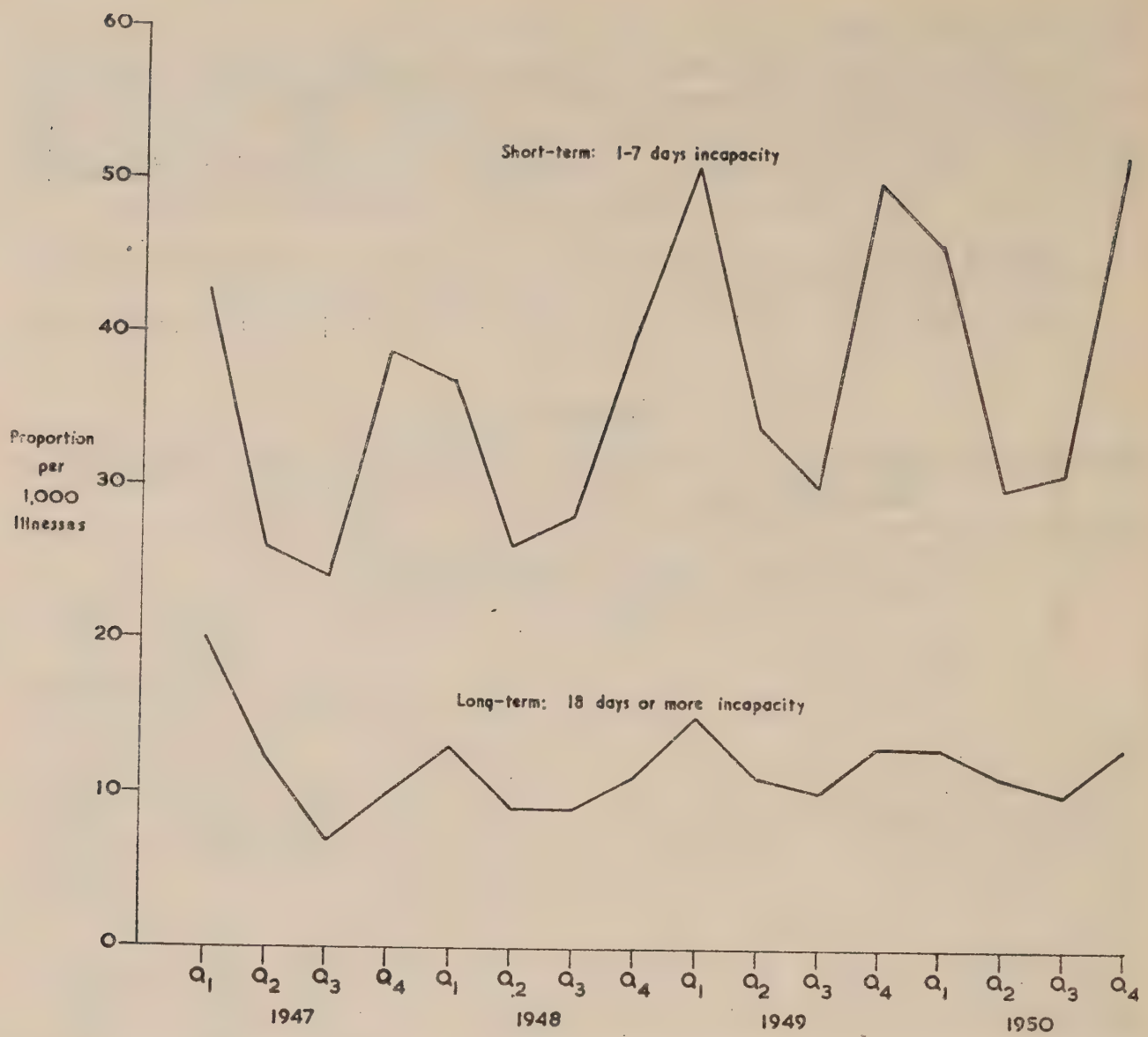


FIG. 2.—Seasonal fluctuation in proportion of shorter illnesses (1–7 days incapacity) and long term (2½ weeks incapacity).

Fig. 2 shows the seasonal fluctuation in the proportions of shorter term illnesses (1-7 days) and longer term (2½ weeks or more).

Types of Illness

Table 4 shows the proportionate distribution of illnesses according to the amount of incapacity caused, for four common types of complaint. Rheumatism excludes chronic rheumatic heart disease, sore throat includes tonsillitis, while among the common ill-defined symptoms are cough, pain in the chest or limbs, nausea and vomiting, diarrhoea, headache and undue fatigue.

TABLE 4

Proportionate Distribution of certain illnesses according to days of incapacity caused and proportionate contribution to total incapacity of illnesses of different durations. Persons aged 16 and over.

Illness	Number of Illnesses per 1,000 causing the following number of days incapacity per year							Percentage of total days of incapacity due to illnesses lasting				
	0	1–3	4–6	7	8–10	11 and over	Total	1–3	4–6	7	8 and over	Total
Rheumatism:												
1947 ...	970	6	5	6	1	12	1,000	4	6	11	79	100
1948 ...	972	7	3	4	2	12	1,000	4	5	7	84	100
1949 ...	957	9	6	5	5	18	1,000	4	6	7	83	100
1950 ...	965	6	5	6	4	14	1,000	4	6	10	80	100
Colds and influenza:												
1947 ...	833	66	26	32	14	29	1,000	12	11	19	58	100
1948 ...	873	62	21	21	8	15	1,000	18	14	21	47	100
1949 ...	839	66	25	27	11	32	1,000	12	11	17	60	100
1950 ...	825	76	27	28	12	32	1,000	13	11	18	58	100
Sore throat:												
1947 ...	743	65	37	58	26	71	1,000	7	8	19	66	100
1948 ...	718	69	49	67	32	65	1,000	7	10	22	61	100
1949 ...	723	77	44	57	25	74	1,000	7	9	18	66	100
1950 ...	685	89	44	78	32	72	1,000	8	9	23	60	100
Ill-defined symptoms:												
1947 ...	969	15	3	3	2	8	1,000	10	6	9	75	100
1948 ...	969	16	4	3	1	7	1,000	13	9	10	68	100
1949 ...	955	14	8	5	5	13	1,000	7	9	9	75	100
1950 ...	963	13	6	4	4	10	1,000	7	8	9	76	100
All illnesses and injuries:												
1947 ...	941	18	7	9	4	21	1,000	6	6	9	79	100
1948 ...	945	18	7	8	4	18	1,000	7	6	10	77	100
1949 ...	927	21	11	10	7	24	1,000	6	7	10	77	100
1950 ...	931	21	9	10	6	23	1,000	6	7	10	77	100

The proportion of illnesses not causing incapacity was highest in each year for rheumatic diseases and lowest for sore throat; these two conditions also had a peak number of illnesses causing 7 days incapacity. For colds and influenza two days was the commonest period of absence and for ill-defined symptoms one or two days. The proportion of sore throats causing incapacity of more than a week and a half was much greater than for other conditions. Of the total amount of incapacity caused by rheumatic diseases four-fifths was due to illnesses of over a week, compared with about three-fifths in the case of sore throats and a little more than half for colds and influenza.

The following summary shows how illnesses of varying lengths of incapacity were distributed per cent. of total illnesses, per cent. of total days of incapacity and per cent. of persons interviewed, monthly averages having been taken for the four years:

Periods of incapacity	Illnesses or Injuries	Total days of Incapacity	Persons interviewed
	per cent.	per cent.	per cent.
0 days	93	0	91
1–3 days	2	6	3
4–6 days	1	7	1
7 days	1	10	1
8 days or more	3	77	4
Total	100	100	100

Some defects of Sickness Absence Data

The above figures, derived from the Survey of Sickness, have certain defects as measures either of the incidence of sickness or of incapacity caused by sickness. Other sources of data about incapacitating sickness also have their defects, primarily because of the difficulties of ensuring that absence from work attributed to sickness is really due to sickness, of allowing for justifiable variations in the reaction to sickness due to variations in working conditions and of allowing for variations in the effects of absence on income. Figures derived from sickness absence records must therefore be interpreted cautiously when attempting to measure the real incidence of sickness.

The study of incapacitating sickness is, however, important in relation to improvement of working conditions and finding the right job for the person and the right person for the job. It can thus contribute to the health and happiness of the worker and to his productivity. There is therefore a need for further examination of the factors involved in absence attributed to sickness, and a comparison of data derived from different sources may provide valuable clues to their extent and influence.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, SEPTEMBER, 1952

Issued from the General Register Office, Somerset House, W.C.2

	September 6th	September 13th	September 20th	September 27th	Average weekly figures, September, 1951
Scarlet Fever	557	701	852	1,164	584
Whooping Cough	1,831	1,487	1,366	1,258	2,338
Diphtheria	23	26	21	15	33
Measles, excluding Rubella ...	2,937	2,421	2,594	3,272	1,361
Acute Pneumonia	228	210	261	316	233
Meningococcal Infection ...	28	26	30	21	25
Acute Poliomyelitis (Paralytic)...	115	116	127	104	47
,, ,, (Non-paralytic)	73	68	62	57	44
Ophthalmia Neonatorum ...	55	29	31	44	33
Puerperal Pyrexia and Puerperal Sepsis	260	203	255	249	250
Dysentery	132	100	103	123	127
Paratyphoid	41	64	33	31	34
Typhoid	7	9	5	3	6
Smallpox	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Sheffield Laboratory : Change of Telephone Number

The telephone number of the Public Health Laboratory at Sheffield is now: Sheffield 36253.

AN EXPLOSIVE OUTBREAK OF SALMONELLA TYPHI-MURIUM FOOD POISONING IN LLANELLY

W. Kwantes, M.A., M.B., B.Chir., Dip.Bact.,
Public Health Laboratory, Carmarthen

During August, 1949, an explosive outbreak of food poisoning caused by *Salmonella typhi-murium* affected several hundred persons in Llanelly, and resulted in infectious cases being scattered throughout the town to such an extent as to interfere with its normal routine. The outbreak undoubtedly originated from a bakery, which on the morning of Saturday, 13th August, sold 720 cream pastries, whose synthetic cream filling was infected with *Salmonella typhi-murium*. The outbreak was brought to the notice of the Health Authorities early on Monday, 15th August, because of the large number of persons who had been taken ill. It soon became apparent that all of those affected had eaten pastries from the bakery in question. Fortunately, two of the households had left over some pastries which were purchased on the previous Saturday. There were six of these pastries, and the synthetic cream in each of them, on bacteriological examination, gave an abundant growth of *Salmonella typhi-murium*. Additional evidence that the cream was the cause of the outbreak was the observation of a mother who infected her seven-month-old infant by giving it a small amount of cream from one of the pastries.

Notification

During the three weeks following the sale of the infected pastries 224 persons were notified as suffering from food poisoning. Table I shows their distribution together with the number who definitely consumed cream pastries purchased from the bakery concerned on 13th August.

TABLE I

Distribution of times of notification of cases

Day following sale of pastries	No. of cases of food poisoning notified	No. who consumed pastries
1	0	0
2	6	6
3	56	56
4	41	40
5	45	42
6	40	37
7	7	7
8–14	22	14
15–21	7	1
	224	203

The close association between the notifications and the consumption of the pastries when taken in conjunction with the previous findings leaves no doubt that these were the vehicle of infection.

Incidentally these figures show the very real delay in the notification of food poisoning, as only half the cases had been notified four days after the sale of the pastries and it took a week before the full extent of the outbreak was apparent.

Method of investigation

After each notification a sanitary inspector visited the household of the notified case and made investigations ; he noted on a specially prepared form the name and age of the case and listed the main symptoms. He also noted whether pastries had been consumed, their time of consumption as well as the time of onset of symptoms. In addition a note was made of the names, ages and occupations of all the other members of the household. All food handlers were excluded from work and the sanitary inspector informed the household of the hygienic precautions necessary to prevent spread of the infection. Each member of the household was given a specimen container and a specimen of faeces was requested. This was collected from the house the following day or later. An attempt was made to obtain weekly follow-up specimens from all those whose initial specimen contained *Salmonella typhi-murium,* and to continue them until either two negatives were obtained or there was a refusal to submit specimens. The co-operation of the public was excellent to begin with, but there was an increase in the number of refusals later on.

Age and sex incidence of notified cases

Persons of all ages were infected in this outbreak, the youngest being seven months old and the oldest aged 80 years. The incidence in the sexes up to the age of 20 years was the same, but above this age slightly more females than males were affected. This was probably because more females than males eat cream pastries after this age.

Table II shows the age and sex distribution of the 203 notified cases that consumed cream pastries, together with the total number of persons of each age group in these households.

TABLE II

Age and sex distribution of cases

Age	Male			Female		
	No. in Households	No. of cases	Per cent.	No. in Households	No. of cases	Per cent.
0–4 yr. ...	33	8	24	34	8	24
5–9 ,, ...	17	4	24	22	5	23
10–14 ,, ...	22	7	32	26	6	23
15–19 ,, ...	25	8	32	41	12	29
20–29 ,, ...	74	23	31	89	30	34
30–39 ,, ...	42	8	19	61	18	30
40–49 ,, ...	57	13	23	56	16	28
50–59 ,, ...	35	8	23	47	17	36
60–69 ,, ...	19	6	32	16	2	13
70–79 ,, ...	7	1	14	11	2	18
80–89 ,, ...				3	1	33
Total ...	331	86	26	406	117	29

The table shows that about a quarter of each age group was affected. There was no suggestion that any age group was less susceptible than any other.

Symptoms

As the cases were treated by 15 different general practitioners, it was not considered possible to obtain exact details of their symptoms. An examination of the sanitary inspector's notes showed that nearly all the patients suffered from vomiting, abdominal pain and severe diarrhoea ; a few had headache or backache as well. Twelve were sufficiently ill to require hospital treatment. A man aged 69 died in hospital 6 days after becoming infected, but at the post-mortem his death was shown to be due to coronary thrombosis.

Incubation period

Information was available in 144 cases of the time of consumption of the cream pastries and the time of onset of symptoms. The time of consumption was often given as "tea time" or "supper time" and estimates of the incubation period were therefore made to the nearest four hours.

Table III shows the number affected after varying intervals.

TABLE III

Distribution of incubation periods

Incubation period (hours)	*No. affected*	*Incubation period (hours)*	*No. affected*
0–	2	24–	21
4–	6	28–	15
8–	15	32–	4
12–	15	36–	4
16–	37	48–	2
20–	21	Over 60	2

Two anomalous findings were excluded from Table III. In one case a man had symptoms before eating the pastries and in another the symptoms occurred at the time of eating the pastries ; these occurrences were probably fortuitous. The mean incubation period was 21 hours for those who noticed symptoms within 48 hours. There were two incubation periods of over 60 hours, namely 3 and 4 days. Each occurred in a household where there were others infected, and it is possible that the patients escaped the initial infection and were secondarily infected.

This outbreak shows a very similar distribution of incubation periods to that of an outbreak described by Garrod and McIlroy (1949) in which 136 persons fell ill after eating a pudding infected with *Salmonella typhi-murium*. The authors gave a figure of 23.8 hours for the mean incubation period for those with symptoms within 48 hours.

Table III has been analysed according to the method of Sartwell (1950). Fig. 1 is a graph on probability paper showing the cumulative percentage of cases affected against the logarithm of the incubation period. A straight line has been fitted by inspection and this line crosses the 50 per cent. line at a point represented by a time in hours whose logarithm is 1.26. Taking the antilogarithm, the estimated median incubation period is 18.2 hours. The dispersion factor was obtained by taking the antilogarithm of half the difference between the log times corresponding to a +1 and −1 standard deviation. For practical purposes these deviations are represented by the 16 per cent. and 84 per cent. cumulative frequencies. The logarithms of the times at these points are 1.05 and 1.47 respectively. Half the difference is 0.21 and its antilogarithm 1.62.

The dispersion factor for this outbreak is therefore 1.62.

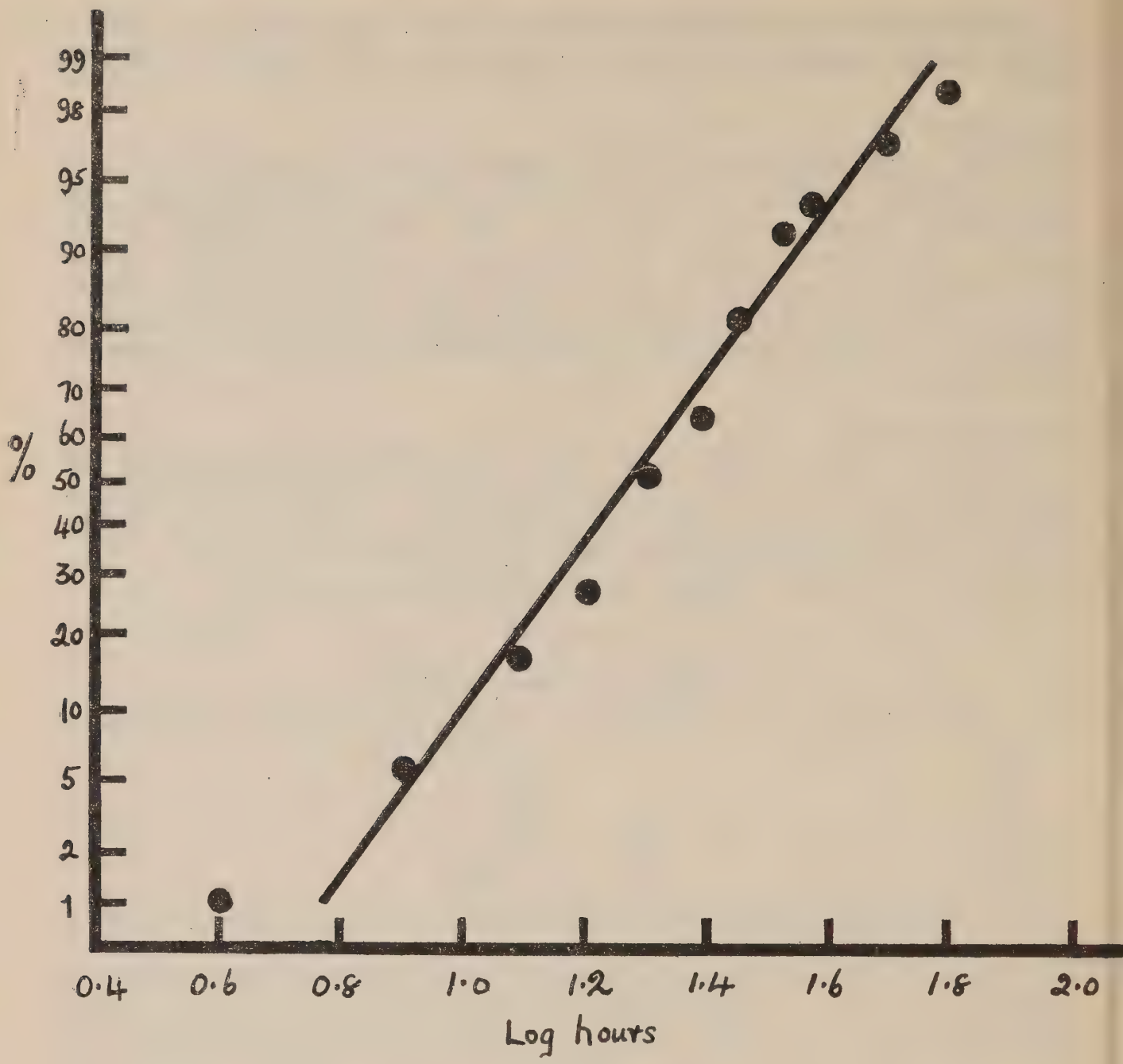


FIG. 1

Graph showing the cumulative percentage of cases affected against the logarithm of the incubation period.

Bacteriology

Specimens of faeces on arrival at the laboratory were plated on to deoxycholate citrate agar and Wilson and Blair's medium, and inoculated into tetrathionate broth. The latter was plated on to deoxycholate citrate agar after 24 hours' incubation at 37°C. In order to economize in medium, two specimens of faeces were streaked on each plate. Deoxycholate citrate agar plates were incubated for 24 hours at 37°C., after which they were examined. Wilson and Blair's medium was examined after 24 and 48 hours' incubation at 37°C. All suspicious colonies were picked on to MacConkey agar plates, which were examined after 24 hours' incubation at 37°C.

A slide agglutination test against *Salmonella typhi-murium* O and H was performed on all suspicious cultures. If positive, they were sent to the Regional Laboratory at Cardiff for confirmation. All cultures failing to react by slide agglutination were subcultured on to Christensen's urea medium, and if this was negative were inoculated into a set of sugars. No pathogen other than *Salmonella typhi-murium* was isolated.

Three strains of *Salmonella typhi-murium*, one from the synthetic cream filling of an éclair and two from cases, were examined at the Central Enteric Reference Laboratory and found to belong to Vi-phage Type 4. The typing of *Salm. typhi-murium* by means of Vi-bacteriophage has been mentioned briefly by Felix and Callow (1943) and Felix (1951), but the provisional typing scheme now in use has not yet been published.

Bacteriological examination of the first specimen of faeces from 224 notified cases gave the following results. Of 203 patients who ate cream pastries, 184 were investigated bacteriologically and *Salmonella typhi-murium* was isolated from 157; of 21 patients who did not eat the pastries, 15 were examined and 4 found to be excreting *Salmonella typhi-murium*. Of the 4 excreting *Salmonella typhi-murium* who did not eat pastries, 3 fell ill four days after the sale of the pastries and could easily have contracted infection from one of the large number of cases or symptomless excreters; the source of their infection was not established. The other was an infant of three months who was ill three days before the sale of the infected pastries.

The failure to isolate *Salmonella typhi-murium* from 27 cases was mainly due to the long interval between the onset of their symptoms and the collection of the specimens.

An analysis of results of the first specimens collected from the 184 cases according to the day of collection is interesting. The results are given in Table IV.

TABLE IV

Results of bacteriological examination of first specimens

Day after the sale of pastries	3	4	5	6	7	9	10	11	12	13	14	Over 14
No. positive ...	13	32	12	35	33	10	13	4	1	3	1	0
No. negative ...	0	0	0	2	2	3	4	2	1	3	0	10

The figures demonstrate the well known fact that the chance of isolating *Salmonella* from cases is high during the first week of infection, but gets progressively less after this.

Duration of excretion

Further specimens of faeces were requested from all cases yielding an initial specimen positive for *Salmonella typhi-murium*. These were asked for at weekly intervals until two negative specimens were obtained. More and more persons refused to provide specimens as time went on, and it was found very difficult to obtain specimens later than six weeks after the original infection. It was however possible to follow up 112 persons. Table V is an analysis of the findings.

TABLE V

Duration of excretion of Salmonella typhi-murium

End of week	Number excreting *Salmonella typhi-murium*	Per cent.
1	112	100·0
2	60	53·5
3	34	30·4
4	16	14·3
5	9	8·0
6	9	8·0
9	At least 3	At least 2·6

The figures show that about a third of the patients were excreting *Salmonella typhi-murium* at the end of 3 weeks, and a small percentage after 9 weeks. The three patients excreting for 9 weeks were aged 21, 55 and 58 years respectively.

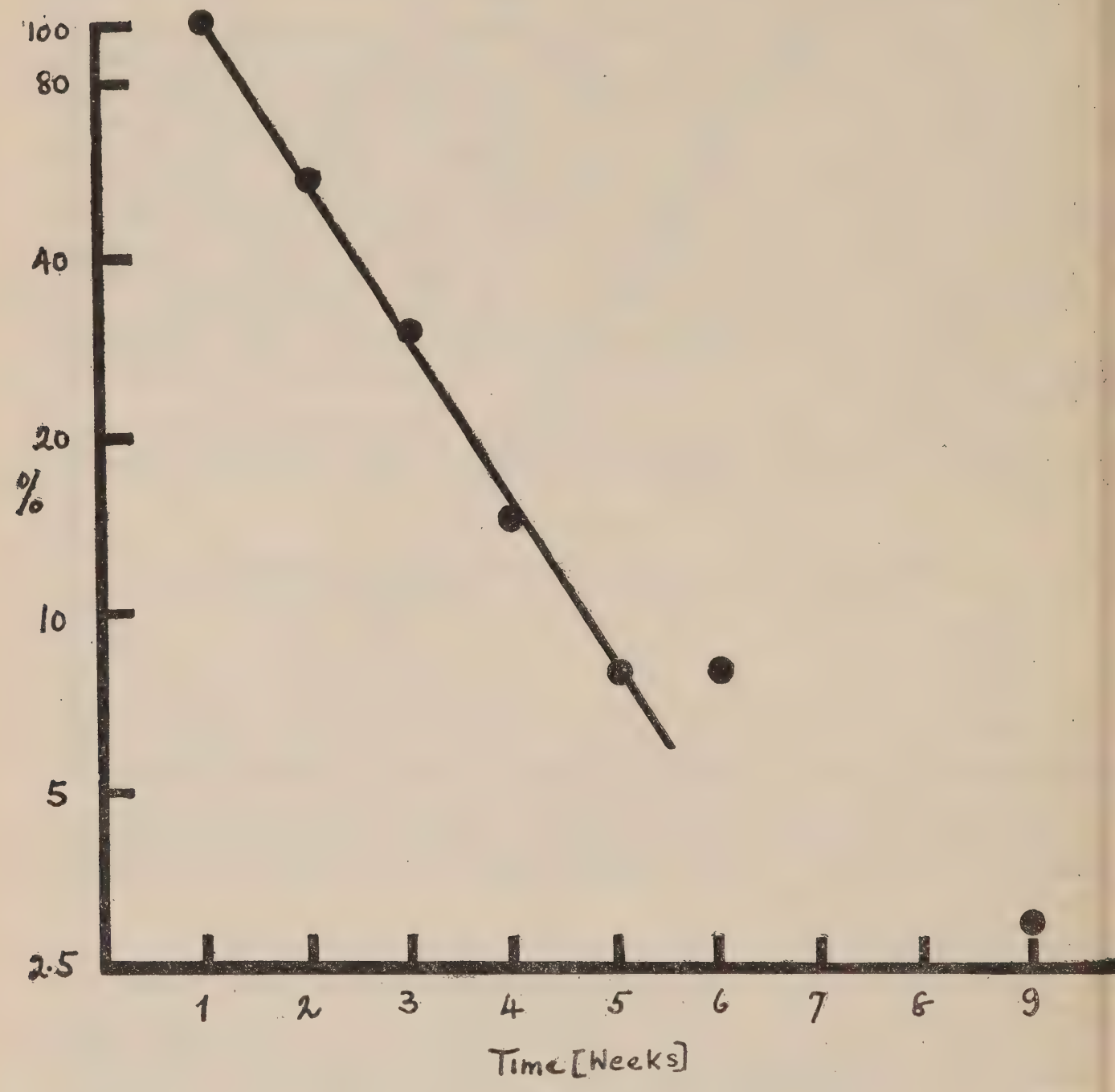


FIG. 2

Graph showing the percentage of persons who continued to excrete *Salm. typhi-murium* in successive weeks (Llanelly outbreak).

Table V has been analysed by plotting on semilogarithmic graph paper the percentage of persons excreting *Salmonella typhi-murium* against time, and the result is shown in Fig. 2. Up to 5 weeks the graph is a straight line but after this there is a slight departure owing to the small number of excreters remaining. The equation $\log n = 2.275 - 0.275t$ gives the calculated percentage n of convalescents still excreting after time t in weeks.

Table VI shows the observed and calculated percentages.

TABLE VI

Duration of excretion: observed and calculated percentages

Weeks	Observed Percentage	Calculated Percentage
1	100·0	100·0
2	53·5	53·1
3	30·4	28·2
4	14·3	15·0
5	8·0	7·9
6	8·0	4·2
9	2·6	0·6

The constant 0.275 in the equation indicates that after the first week about 47 per cent. of convalescents excreting *Salmonella typhi-murium* at the end of any one week will have ceased excreting it by the end of the next week. This is true up to 5 weeks, but after this the figures for the outbreak are too small to check the reliability of the equation.

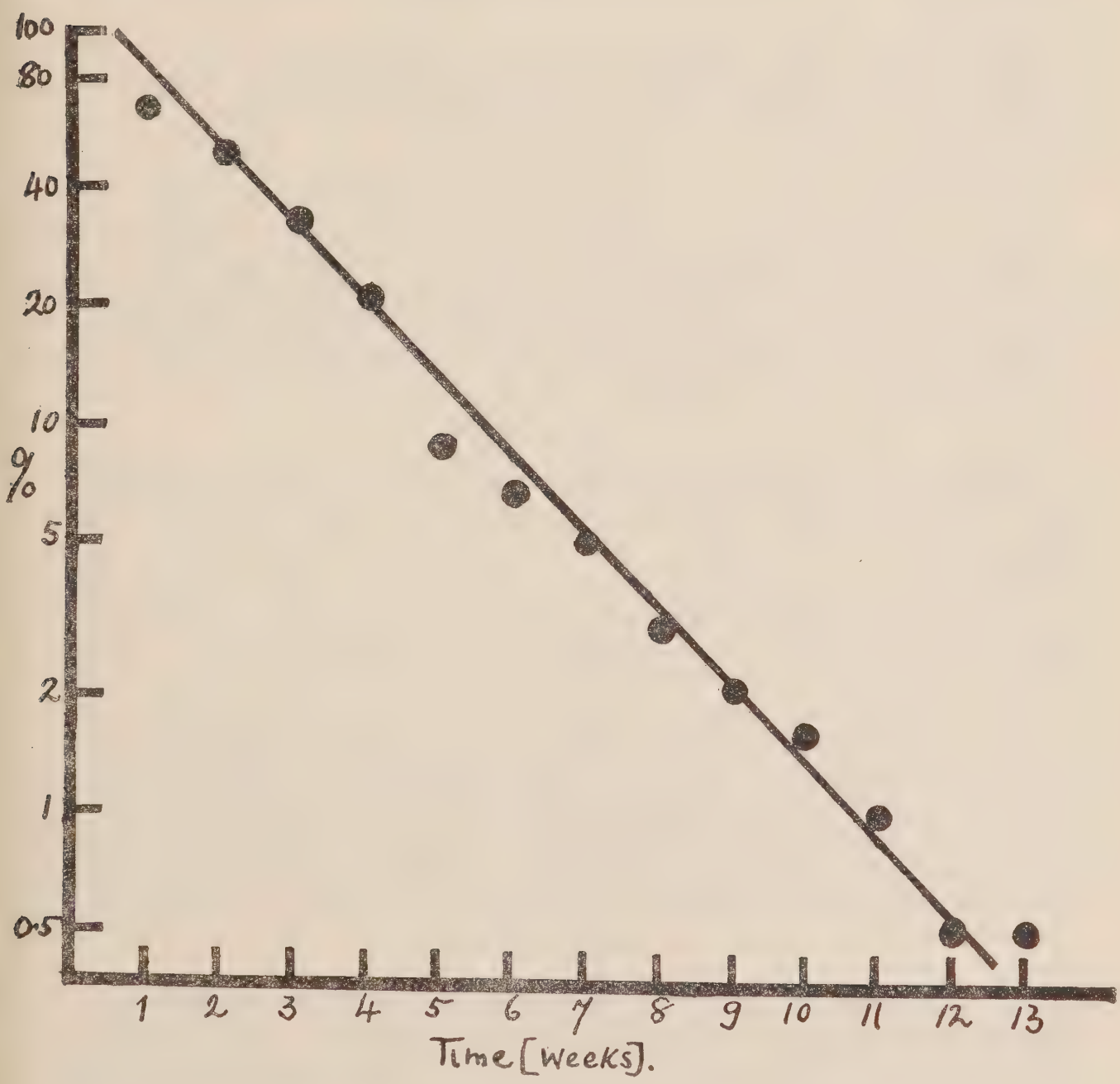


FIG. 3

Graph showing the percentage of persons who continued to excrete *Salm. typhi-murium* in successive weeks (Mosher *et al.*, 1941).

Mosher, Wheeler, Chant and Hardy (1941) followed up over a period of 13 weeks the duration of excretion of *Salmonella typhi-murium* by 195 convalescents in an institution for mental defectives. Their figures plotted on semilogarithmic paper are shown in Fig. 3 and give a straight line for the whole of this period. The clearance rate calculated from this straight line is 39 per cent. per week. It appears probable that a logarithmic law governs the duration of excretion of *Salmonella typhi-murium* in the faeces.

Mild cases

The co-operation of the general practitioners in Llanelly was excellent. A circular was sent to them on the 4th day of the outbreak asking them to notify all cases of suspected food poisoning seen by them. All cases ill enough to be seen by a doctor were notified.

However, in the households of notified cases, the sanitary inspectors discovered a further 8 persons who were ill enough to go to bed for a day or so, but not ill enough to call in a doctor. They had all consumed the infected pastries and all of them were found to be excreting *Salmonella typhi-murium.*

Other cases

Three cases occurred in persons who had visited Llanelly for the day but had left the area. They were all ill enough to go to hospital.

Symptomless excreters

As previously mentioned, specimens of faeces were collected from as many persons as possible in every household of a notified case. Specimens were collected from 357 persons who had either no symptoms at all or only a very minor intestinal disturbance which was insufficient for them either to go to bed or send for the doctor; 94 symptomless excreters were discovered among them. An analysis of the initial specimen of the 357 persons according to the date of collection is given in Table VII.

TABLE VII

Results of bacteriological examination of persons in infected households

Day after sale of pastries ...	3	4	5	6	7	9	10	11	12	13	14	Over 14
Number positive	4	7	2	24	24	8	8	4	1	1	0	8
Number negative	5	7	4	32	45	19	26	23	10	2	1	92

It is interesting to note that during the first week 61 out of 154 persons were found to be symptomless excreters. Reference to Table II shows that there were 737 persons living in the affected households; of these, 211 had definite symptoms leaving 526 presumably healthy persons. If all these persons were affected in the ratio of 61 to 154 this would mean that there were 208 symptomless excreters during the first week of the outbreak. In other words there were nearly as many symptomless excreters as there were cases.

This conclusion is probably justifiable when it is remembered that 720 infected pastries were sold and the sanitary inspectors' notes showed that quite a number of the symptomless excreters ate these infected pastries. Symptomless excreters were found at all ages. The youngest was 10 days old and the oldest 80 years old; three were under six months. These were most probably secondarily infected, as they are unlikely to have eaten the pastries.

Duration of excretion of symptomless excreters

It was found possible to follow up 36 of the 61 symptomless excreters who had a positive specimen during the first week of the outbreak (Table VIII).

TABLE VIII

Duration of excretion of Salmonella typhi-murium in symptomless excreters

End of week	Number excreting *Salm. typhi-murium*	Per cent. excreting *Salm. typhi-murium*
1	35	97
2	13	36
3	11	31
4	6	17
5	2	5·5
6	2	5·5
7	2	5·5

This table shows that the duration of excretion of *Salm. typhi-murium* by symptomless excreters was not grossly different from that of cases and that symptomless excretion may continue for several weeks. The two persons who excreted *Salm. typhi-murium* for seven weeks were aged 8 months and 6½ years respectively. The three infants who were under six months of age excreted for 4, 4 and 5 weeks respectively.

Method of infection of the cream pastries

The synthetic cream used for the filling of the pastries was obtained in 1 gallon quantities once a fortnight from a reputable commercial firm. A tin was opened at 7.30 a.m. on the morning of Saturday, 13th August, and put into a large bowl with about 1 lb. sugar and the white of 10 eggs, and the whole mixed with a mechanical mixer. The cream was then dispensed into the pastries, which were all sold over the counter between 9 a.m. and 1 p.m. The shop was first visited by the public health authorities on Monday, 15th August, and by that time the tin which contained the synthetic cream had been thrown away. Specimens of faeces were requested from the eight members working in the bakery, and six of them were found to contain *Salm. typhi-murium*. One of the excreters had taken home some of the cream pastries on the Saturday; both she and her sister ate them for tea and had symptoms of infection the following day. The remaining five were symptomless excreters, two of whom were concerned in making the pastries. The possibility exists that one of these two infected the pastries. There is another possibility, namely that the cream mix was infected by the egg albumen. Both hens' and ducks' eggs were received by the establishment, and duck egg albumen infected with *Salm. typhi-murium* may have been used in the synthetic cream mixture. It was not possible to trace the source of the duck eggs used for the pastries as they were taken from a pool of 673 eggs collected from 48 different farms during the week before the outbreak.

Summary

An explosive outbreak of food poisoning due to *Salmonella typhi-murium*, Vi-phage Type 4, affecting several hundred persons in Llanelly in 1949 is described. The food causing the outbreak was the synthetic cream filling of cream pastries sold by a bakehouse.

Statistical analysis of the incubation periods of 144 cases showed that they followed a logarithmic law and gave a median incubation period of 18.2 hours and a dispersion factor of 1.62.

Salmonella typhi-murium was isolated by stool culture from 169 of the cases, and 94 symptomless excreters were discovered in this way. A hundred and twelve of the cases were followed up bacteriologically for several weeks. The percentage of persons still excreting *Salmonella typhi-murium* after various intervals was found to follow a logarithmic law ; 47 per cent. of those excreting *Salmonella typhi-murium* at the end of one week had ceased excreting the organism by the end of the following week.

The possible origin of the outbreak is discussed.

I wish to acknowledge gratefully the co-operation given by Dr. Rees Evans, the County Medical Officer, and his deputy, Dr. D. G. G. Jones, Dr. D. Vernon John, the acting Medical Officer of Health of Llanelly Borough, and especially the sanitary inspectors and staff of the Llanelly Borough and Rural Public Health Departments, who made hundreds of visits to the homes of cases ; also to Dr. Harvey at the Public Health Laboratory, Cardiff, for the confirmation of the *Salmonella* strains, and Mr. M. J. Anderson, A.I.M.L.T. for drawing Figs. 1, 2 and 3.

References

Felix, A. (1951) *Brit. med. Bull.*, **7**, 153.

Felix, A. and Callow, B. R. (1943) *Brit. med. J.*, ii, 127.

Garrod, L. P. and McIlroy, M.B. (1949) *Brit. med. J.*, ii, 1259.

Mosher, W. E., Wheeler, S. M., Chant, H. L. and Hardy, A. V. (1941) *Publ. Hlth Rep., Wash.*, **56**, 2415.

Sartwell, P. E. (1950) *Amer. J. Hyg.*, **51**, 310.

Editorial Matter for

I.—The GENERAL SECTION to

Editor,
Ministry of Health (Room 207),
Savile Row,
London, W.1.

Tel.: REGENT 8411. Extn. 91.

II.—LABORATORY SECTION

Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.

Tel.: WHITEHALL 4884.

The September Bulletin was issued on 23rd September.

Note.
Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(17103) Wt. 8221—7283 3,300 10/52 D.L.

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

NOVEMBER **1952** **Vol. 11**

CONTENTS

Section I

Section II

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, London, W.1.

WINTER EPIDEMICS

In December, 1951, a note on Winter Epidemics was published in the monthly bulletin of the Ministry of Health and P.H.L.S. drawing the attention of medical officers of health and medical practitioners to the present state of our knowledge of respiratory infection of presumed virus aetiology. It was indicated that there was a desire to make a renewed effort to investigate the problem in the field and in the laboratory in this country with the aid of the public health and practitioner services. One of the main lines of approach was to be the attempt to correlate laboratory findings (particularly those for influenza viruses) with clinical diagnoses and with local increases in morbidity as evidenced by increases in the numbers of fresh claims to sickness benefit under the National Insurance scheme. The eventual aim is also to determine the virus involved at the beginning of an epidemic, investigate the possibility of using vaccine and find out more about the epidemiology of virus influenza.

(1) *Development of Laboratory Investigations*

In previous years serological and virological tests for influenza were carried out mainly at Mill Hill (the National Institute for Medical Research), Colindale (the Virus Reference Laboratory), Northampton (Public Health Laboratory) and Sheffield (the Department of Medicine) with the support of certain other university bacteriology departments. In the winter of 1951–52, further facilities for serological diagnosis of influenza were made available in nine other P.H.L.S. Laboratories at Bradford, Cambridge, Cardiff, Exeter, Hull, Manchester, Newcastle, Nottingham and Winchester. Throat washings (garglings) from areas other than Northampton and Sheffield were sent from the above laboratories to the Virus Reference Laboratory, Colindale, for attempted virus isolation in fertile hens' eggs.

(2) *Incidence of Influenza during Winter of* 1951–52

An index of the low incidence of influenza in this country last winter is found in the number of deaths certified as due to that disease: between 25 and 45 deaths a week were so certified in the 160 great towns and this is but little higher than in 1948 which provided the low record.

The effect of the absence of an epidemic on the productivity of the nation is noteworthy. In round figures and by comparison with 1951 about one million fewer persons were incapacitated during January and February, 1952, and there were 50,000 fewer deaths.

Notwithstanding the increased facilities for the laboratory investigation of influenza, Virus A was not identified in England and Wales during the winter. A few outbreaks of Virus B occurred but were strictly circumscribed.

The first was reported from Taunton (Somerset) in late January and involved the patients and staff in one ward of a hospital. Serological evidence of virus B infection was obtained in six out of ten patients in the ward, three of whom showed a rising titre of antibody. These cases arose over a period of twelve days. A little later there was a sharp rise in the number of new claims to sickness benefit in south west England, which might suggest that the Taunton incident was part of a wider spread. However, 94 sera were examined from 57 patients in the area suffering with illnesses considered by their medical practitioners to be typical influenza, and in all of these cases the serological findings were negative for virus A and B. The probability is that some other infection was also rife.

Somewhat later still, at the end of February, virus B was actually recovered from throat washings from a patient in Chard (12 miles from Taunton).

During February, serologically proven infection was also reported from Plymouth, London and Sheffield (and was confirmed in Sheffield where virus B was isolated (Stuart-Harris)).

During March, further isolated outbreaks of virus B infection occurred and were proved serologically in Hull, Leicester, Wakefield, and Bradford, where a strain of virus B was isolated.

In the early part of April no new foci of infection were reported, but proven cases were still occurring in the London and Bradford areas.

In addition, the presence of high titres of antibody in single (convalescent) specimens of serum from a few patients suggested that influenza B infection was also present in Bath, Cambridge, Leeds and Nottingham, in the period between the end of January and the beginning of April.

The following table details the specimens received by the P.H.L.S. from January to April, 1952, inclusive. This is, of course, partially an index of the incidence of influenza-like illness, but mainly a reflection of the interest shewn by the practitioners and medical officers of the region. No conclusions as to the presence of influenza virus B can be drawn except in areas where positive results were obtained.

Laboratory	Specimens received (persons)		
	Serum	Throat Washings	P.M. Tissue
Bradford	12	12	2
Cambridge	15	4	0
Cardiff	30	1	3
Colindale	250	45	6
Exeter	37	37	0
Hull	167	0	0
Manchester	10	0	0
Newcastle	4	1	0
Northampton	15	3	0
Nottingham	8	0	0
Winchester	4	0	0
TOTAL	552	103	11

Although only two virus isolations were made from the throat washings examined at Colindale and 101 garglings were negative, eleven of these negative patients were proved to have had influenza B by showing a rising titre of antibodies. Negative results were obtained in the following cities:—

Cardiff (30 sera, 1 gargling and 3 lungs); Northampton (15 sera, 3 garglings) and Manchester (10 sera and no garglings); but the numbers are too small to be of any significance.

(3) *Vaccine Trials*

The low incidence of influenza permitted a controlled investigation of the laboratory evidence (as judged by a rise in antibody titre) of the relative merits of selected influenza vaccines. As a result of this study, it has now been possible for the M.R.C. Influenza Vaccine Trials Committee to make arrangements for a controlled field trial of the vaccine of choice under whatever epidemic conditions the winter of 1952–53 may hold in store. The extent of this trial is of necessity limited, and a separate communication will therefore be addressed to those medical officers of health more immediately concerned.

The interest of the Ministry and laboratory medical staff is by no means limited to the areas involved in the vaccine trial. The necessity still remains for the sampling of any outbreak of acute respiratory infection in whatever area it may occur, and in particular of suspected influenza in the early part of the season. Further, it is essential to catch the individual case at the onset in order to obtain the most suitable specimens for the isolation of virus. In this connection it has also to be borne in mind that it is only from material obtained at the optimum time (early in the illness) that virus is likely to be recovered and it is only by a constant study of the prevalent type of virus that any necessary modifications can be made in the vaccine.

(4) *Outlook for* 1952–53

The relative freedom from virus influenza enjoyed by this country last winter was shared by the rest of Europe and by North America. It is of interest that up to the end of August, 1952, the presence of outbreaks of Virus A, so widespread a year previously, had not been reported from either of the above continents, in both of which special arrangements had been made for the early reporting and laboratory investigation of influenza-like outbreaks. The presence of Virus A was, however, reported from South Africa in June of this year, i.e. the South African winter. If events in the coming winter follow the experience of two years ago, the appearance of influenza Virus A in South Africa in June may again be followed by its occurrence in Northern Europe six months later, in our winter. Should this mature—and it is indeed in keeping with the biennial cycle of Virus A epidemics which has been a feature in recent years—public health departments will want to keep themselves informed of the position locally and it is also hoped that medical officers of health will continue to co-operate with the Ministry's Epidemiological Section and the laboratories.

(5) *Ascertainment and investigation*

The suggestions made a year ago in the December issue of this bulletin still apply. They were as follows:—

Clinical appraisement of an epidemic rests mainly with general practitioners and the medical staffs of hospitals, and it is suggested that medical officers of health should make arrangements now with practitioners who would act as "spotters" and furnish regular reports when the occasion arises.

The immediate effect of an epidemic on industry and on residential communities is an increase in sickness and absence from work. Arrangements might be made with the medical officers concerned for reporting to the medical officer of health any significant increase in upper respiratory illness in the staff or community.

The further effect of an epidemic is next shown by a rise in new claims to sickness benefit, and it is suggested that medical officers of health renew or reaffirm arrangements made previously with local officers of the Ministry of National Insurance for immediate intimation of any sharp local rise in these claims. The general value in epidemiology of studies of the variations in first sickness benefit claim figures is brought out in the 1951 Annual Report of the Ministry of National Insurance (published in August, 1952) which contains an account of the large 1950–51 outbreak treated from that angle. Arrangements should also be made with local registrars of death to provide daily reports on total deaths and deaths from pneumonia, bronchitis and influenza during the period of an epidemic.

The information received through such channels will indicate the presence and extent of any epidemic of acute upper respiratory illness, but the differential diagnosis of epidemic influenza from other clinically similar illnesses is largely a laboratory procedure. It is therefore requested that the medical officer of health should now discuss with the directors of public health laboratories or, in some instances, hospital laboratories, the mechanism for bringing to the notice of the laboratory the very first indications of an outbreak and for providing the laboratory with the special material required. For this the medical officer of health may wish to bring the laboratory into direct contact with the general practitioners acting as " spotters " so that the necessary collecting outfits may be provided before the epidemic begins.

When the medical officer of health decides that an unusual prevalence is to be expected, a note giving the information available should be sent to S.M.O., Med. 3, Ministry of Health, Russell Square, W.C.1. This preliminary notice, describing the outbreak in general terms will be of great value to the Ministry and it is hoped that most medical officers will follow this up by sending reports for weeks ending on Saturdays, should the outbreak be substantial.

In the case of ports and airports, port medical officers are particularly requested to report immediately to S.M.O., Med. 3, Ministry of Health, the occurrence of influenza-like disease aboard ship or amongst passengers or aircrew arriving in this country. It is hoped that similar reports will be made by the armed service medical authorities concerned in the case of aerodromes and ports under service control. Arrangements for investigation will be made according to the circumstances.

The experience in South West England at the beginning of 1952 is an example of the difficulties which have to be overcome. The solution probably lies in more intensive clinical and epidemiological studies in the immediate locality of outbreaks.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, OCTOBER, 1952

Issued from the General Register Office, Somerset House, W.C.2

	October 4th	October 11th	October 18th	October 25th	Average weekly figures, October, 1951
Scarlet Fever	1,284	1,617	1,760	1,994	1,038
Whooping Cough	1,126	1,078	1,074	1,205	1,540
Diphtheria	25	24	29	28	35
Measles, excluding Rubella ...	4,696	5,716	7,785	9,178	1,722
Acute Pneumonia	371	416	439	519	387
Meningococcal Infection ...	29	40	30	21	34
Acute Poliomyelitis (Paralytic)...	101	92	86	75	60
,, ,, (Non-paralytic)	54	17	33	26	42
Ophthalmia Neonatorum ...	32	28	29	29	38
Puerperal Pyrexia and Puerperal Sepsis	219	256	254	251	236
Dysentery	192	160	181	236	139
Paratyphoid	23	25	15	8	58
Typhoid	4	2	6	8	6
Smallpox	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

Bath Laboratory

The Public Health Laboratory Service have established a new laboratory at the Manor Hospital, Combe Park, Bath (*Tel:* Bath 7651/2). The Director is Dr. P. G. Mann.

Dysentery Reference Laboratory: Change of Address

The Dysentery Reference Laboratory has moved from Oxford, and is now housed in the Central Public Health Laboratory, Colindale Avenue, London, N.W.9 (*Tel.:* Colindale 7041).

FILTER PAPERS AND THE PHOSPHATASE TEST

G. T. Cook, M.D., Public Health Laboratory, Oxford, and
K. E. A. Hughes, M.B.E., M.R.C.S., L.R.C.P., Public Health Laboratory, Portsmouth

Under the Milk (Special Designation) Regulations, 1949, pasteurized milk is required to satisfy the phosphatase test. Details of the method of carrying out the test are contained in the regulations, which include a list of precautions to be observed for the avoidance of false results. The test is deemed to be satisfied by milk which gives a reading of 2·3 Lovibond blue units or less. Two control tests are included: (1) The "milk control" consists of milk that has been added to the buffer-substrate and test reagent but not incubated, the test being then completed in the usual way. If the colour reading exceeds 1·5 units, the test is void. (2) The "reagent control" test is made by incubating the buffer-substrate and chloroform without milk and completing the test as usual. The reading must not exceed 0·5 unit.

Observations at Portsmouth, Winchester and Oxford

During February, 1951, difficulties were encountered with the phosphatase test at the Public Health Laboratory, Portsmouth. An unexpectedly large proportion of the routine milk samples gave high readings and apparently failed. However, as many of the milk controls and the reagent controls gave readings above 1·5 and 0·5 Lovibond blue units respectively, the tests were invalidated. For example, out of 18 tests put up on 8th February, 5 failed, each giving readings of 3 blue units. The milk controls of these 5 showed figures of from just above 1·5 up to 2·3 blue units. Eight other milks just passed the test with 2·3 blue units each: the remainder passed satisfactorily with readings of 2·0 or less. The reagent control for this batch was about 1·0 blue unit and of a rather more greenish tint than usual. During the following two weeks 44 more tests were performed with 8 apparent failures; six of these had milk controls above 1·5, one exactly 1·5 and one below 1·5 blue units. The reagent controls were again high. Of the remaining 36 milks, many only just passed the test with readings of 2·3 instead of being below 2·0 blue units, as more frequently happens. The reagents used were examined by serial elimination and replacement in the test. Only the filter papers, double acid-washed Grade A, appeared to be at fault, as certain milks failed after filtration through them and passed after filtration through papers of another batch.

The Public Health Laboratory at Winchester, which also experienced similar difficulties about the same time and traced the fault to the Grade A filter papers, carried out the following experiment:

> A filter paper was soaked in buffer-substrate for 22 hours at 37°C. and the phosphatase reaction carried out on the resultant fluid. This was repeated on a second paper from another box of the same batch. In all, four different batches and eight boxes were tested. Milk control tests using boiled milk were also put up and these were filtered through papers from the same eight boxes.
>
> The results are shown in Table 1.

TABLE 1

Results in blue units of extraction and filtration tests on four batches of Grade A filter papers

Batch No.	Extract in buffer substrate	Milk Control
Satisfactory paper in current use	1·0	less than 1·5
	1·0	1·5
562/3	3·0	2·1
	2·5	1·8
589	4·0	1·8
	2·5	1·8
604/5	2·3	2·1
	2·5	2·1

It was obvious from these results that something in the filter papers was interfering with the test. Six of the eight milk controls showed figures above 1·5 blue units and soaking tests on the same six papers gave readings from 2·3 blue units upwards. Experiments carried out in the Portsmouth Laboratory gave results corresponding to those obtained at Winchester.

Similar difficulties with the phosphatase test had previously been experienced by the Oxford Public Health Laboratory in June, 1948, and had been traced to the batch of filter papers in use at that time. These were Grade B—a double acid-washed paper with a slower filtration time than Grade A. Reagent control tests set up without incubation and filtered through these old papers gave readings of approximately 1·5 blue units, though similar tests made with Grade B papers of a new batch gave satisfactory results. Three samples of pasteurized milk tested in duplicate gave readings of 2·5—3·0 blue units when filtered through the old papers compared with 1·8—2·3 blue units when filtered through paper of the new batch.

Experiments were carried out at Oxford by Dr. R. D. Gray to determine to what extent the reacting substance present in the filter papers could be extracted by prolonged contact with distilled water. Four filter papers of each batch were mashed up in 25 ml. of warm distilled water and retained in the incubator at 37°C. for 15 minutes. Approximately half of the fluid was then removed for test; the remainder was left in the incubator for 20 hours. Table 2 shows the series of control tests set up and the results.

TABLE 2

Investigation of Grade B Filter Papers by Control Tests employing extraction and/or filtration

Series No.	Control Tests	Results in Blue Units with filter papers	
		Old	New
1	10·5 ml. distilled water (filtered).	1·3	0·5
2	10·5 ml. ¼-hour extract (filtered).	3·5	1·0
3	10 ml. distilled water + 0·5 ml. ¼-hour extract (unfiltered).	0·5	0·2
4	10 ml. distilled water +0·5 ml. 20-hour extract (unfiltered).	0·5	0·3
5	10 ml. distilled water +0·5 ml. 20-hour extract (filtered).	1·5	0·7

As would be expected the highest figure was obtained when the effects of extraction and filtration were superadded (Series 2–filtration was necessary here to obtain a clear fluid). In this severe test a significant amount of reacting substance was obtained, even from the new filter papers. It is noteworthy that prolonged extraction (for 20 hours) showed no greater effect than that for the shorter period ($\frac{1}{4}$-hour) and that filtration more than doubled the effect of the 1/20 dilution of the 20-hour extract.

At that time no special investigation of Grade A filter papers was undertaken, but since 1948 these have replaced the Grade B papers previously used at the Oxford laboratory and have proved satisfactory, though the reagent control usually gives a reading of about 0·5 blue unit. In March, 1952, however, a batch of Grade A papers was associated with high readings and the apparent failure of one milk, though both the milk and reagent control also failed. Readings were within normal limits for the milk and milk control when these were centrifuged instead of filtered, and for the reagent control if unfiltered.

As the experiments of 1948 had shown that a reacting substance was obtained even from the new batch of filter papers, it was decided to examine several unopened boxes of different batches. These were taken direct from the store where there was no possibility of contact with phenolic substances. A filtered reagent control without pre-incubation was put up in duplicate for each batch. Two additional reagent control tubes were included and left unfiltered. In all, nine different batches were examined in this way—six batches of Grade A papers and one batch each of Grade B, Grade C (single acid-washed) and Grade D (unwashed) papers. Reagent control tests that included filtration through these papers all gave readings of approximately 0·5 blue unit, three (including two of those filtered through Grade A papers) being slightly above this figure and two slightly below it. The unfiltered control tubes gave a reading of just above 0. It was thus apparent that papers of nine different batches and of four different grades contained a substance capable of reacting with Folin and Ciocalteu's reagent and imparting to the reagent control a blue colour which approximated to 0·5 blue unit and exceeded it in three of the nine tests.

In experiments undertaken to determine whether faulty papers could be rendered satisfactory by washing, it was found that a reading greater than 0·5 blue unit given by a reagent control after filtration through a Grade A paper was reduced to less than 0·5 if the paper was first washed with 12 ml. of distilled water. The washings contained a significant amount of reacting substance and gave a reading of nearly 0·5 blue unit.

Discussion

Many factors have been held responsible for affecting the reliability of the phosphatase test. The Milk (Special Designation) (Pasteurized and Sterilized Milk) Regulations, 1949, stress the danger of contamination of apparatus and reagents with phenolic compounds. It would now seem that filter papers used in the phosphatase test may contain a substance which reacts with Folin and Ciocalteu's reagent to produce a blue colour. For most of the batches tested the amount of this reacting substance present in any individual paper was not sufficient to give a reagent control reading above 0·5 blue unit and did not interfere with the test. These papers gave a buff colour or " negative " result with the spot test described below. From an occasional batch, however, single papers may contain sufficient reacting substance to produce so deep a colour that many pasteurized milks may fail to satisfy the phosphatase test, and the readings of the milk and reagent controls may exceed the permitted levels. Under these conditions the whole test is invalidated.

We do not know the nature of this reacting substance, though it may possibly be some phenolic compound. The substance has been detected in filter papers from unopened boxes which had been kept in a store where there were no chemicals. There is, however, no information about storage conditions before the papers arrive at the laboratory store. The reacting substance can be at any rate partly removed from the filter papers by washing them with distilled water immediately before use and its presence can be demonstrated in the washings. If the substance is present during manufacture, the process of acid-washing appears to have little or no effect on the amount left in the paper, as control tests including filtration through unwashed and single-washed papers gave readings similar to those obtained with double acid-washed papers.

When difficulty arose at the Portsmouth laboratory it was at first thought that the incriminated papers might have absorbed phenol from the air, as carbol-fuchsin staining was carried out close by. The papers were therefore tested for traces of phenolic compounds by the spot test described by Bray, Thorpe and White (1950).

The detecting agent is an alkaline solution of diazotized *p*-nitraniline, prepared as follows:—

(*a*) *p*-nitraniline 0·3 per cent. in 8 per cent. (w/v) HCl ... 25·0 ml.
Sodium nitrite 5 per cent. (w/v) ... ... 1·5 ml.
mixed immediately before use.

(*b*) Sodium carbonate (Na_2CO_3) 20 per cent.

The filter papers are sprayed with solution (*a*) followed immediately by solution (*b*). Some phenolic compounds produce a reddish colour, the rapid development of which indicates that the paper is unsuitable for the phosphatase test. The appearance of a buff colour can be ignored.

A strong positive reaction was given by the incriminated papers and also by papers from a proportion of fresh unopened boxes obtained from the store where there was little chance of their having been contaminated. Milks and reagent controls filtered through these papers as in the phosphatase test gave a high reading, whereas papers negative to the spot test gave satisfactory results. All new boxes of papers at the Portsmouth laboratory are now spot-tested before being accepted for use, but no further unsatisfactory batches have been encountered.

At the Oxford Public Health Laboratory new boxes of filter papers are screened by setting up without previous incubation two reagent controls, only one of which is filtered. This takes very little time and has the advantage that the results are read in the same way as in the actual test. It is useful to include in any series of milks put up for the phosphatase test a second reagent control which will not be subjected to filtration. This will act as an indicator of any significant defect of the filter paper which may have arisen since the box was opened.

Summary

Some batches of filter papers used for the phosphatase test have been found to contain a reacting substance in sufficient quantity to cause a properly pasteurized milk filtered through such a paper apparently to fail to satisfy the test. These papers also give rise to milk control and reagent control readings higher than the permitted figures of 1·5 and 0·5 blue unit respectively.

The reacting substance was present in most other batches of filter papers tested, though not in sufficient quantity to give a reagent control reading above 0·5 blue unit.

Methods of screening new batches of filter papers are suggested.

Our thanks are due to the staff of the Winchester Public Health Laboratory for their co-operation during part of this investigation, and to Mr. J. Durrant, B.Sc., of the Central Laboratory, Portsmouth, for his assistance with the spot test.

Reference

Bray, H. G., Thorpe, W. V. and White, K. (1950) *Biochem. J.*, **46**, 271.

POST SCRIPTUM.—From information obtained from the suppliers since this paper was written, it would appear that, with one exception, all the papers giving high reagent control readings, *i.e.* containing abnormal amounts of the reacting substances, were manufactured over a period of 11 days at the end of April and beginning of May, 1951. It is possible, therefore, that the fault was due to the accidental contamination or to some aberration of the processing, of the papers during this period rather than to any inherent fault in the normal method of manufacture.

Though we have not mentioned the specification of the papers examined, we should like to take this opportunity of thanking the manufacturers, Messrs. W. and R. Balston, Ltd., for their co-operation in endeavouring to find out the cause of the trouble.

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health,
Savile Row (Room 207),
London, W.1.
Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The October Bulletin was issued on 21st October.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired, in case the number is out of print.

(17243) Wt. 8221—7283 3300 11/52 D.L./HE/4

MONTHLY BULLETIN

OF THE

MINISTRY OF HEALTH

AND THE

PUBLIC HEALTH LABORATORY SERVICE

directed by the

MEDICAL RESEARCH COUNCIL

DECEMBER **1952** **Vol. 11**

CONTENTS

Section I

Section II

SECTION I.—GENERAL

Issued from the Office of the Ministry of Health, Savile Row, W.1.

DIPHTHERIA IN DARWEN, 1937–52

R. C. Webster, M.D., D.P.H., D.C.H.
Divisional Medical Officer of Health and School Medical Officer,
Lancashire County Council.

Darwen is an industrial borough in Lancashire, with a census population of 30,827 (Census 1951). There are some 4,000 children in the five to fifteen year age group, and about 1,500 in the one to five year age group. During the years 1937–43 diphtheria was prevalent; the corrected notifications are shown in Table I, and also the deaths for each year, from 1937 to 1950 inclusive.

TABLE I

Year	Notifications	Deaths
1937	61	2
1938	97	3
1939	60	2
1940	19	1
1941	45	3
1942	21	nil
1943	36	3
1944	8	1
1945	2	nil
1946	2	nil
1947	1	nil
1948	1	nil
1949	4	nil
1950	nil	nil

All the cases which occurred from 1939–43 were under my own care, and in 1944–48 under the care of the late Dr. Jane O. Millar.

A case seen in 1941 gave a valuable clue to the outbreak of 1951–52. In 1941 a boy was brought to the school clinic by his mother because she had noticed an alteration in his voice. His intonation was characteristic of palatal paresis, and inspection confirmed this. The mother admitted that some six weeks earlier he had had a sore throat and he was generally "off colour". She scoffed at the suggestion that he had in fact had diphtheria, and refused to have swabs taken. Fortunately he showed no signs of heart involvement, and his voice gradually returned to normal.

In 1937 immunisation of children with a single dose of 0·5 c.c. A.P.T. was begun, and in 1938 two doses, 0·2 c.c. and 0·5 c.c. A.P.T. at an interval of two weeks were used. War came in 1939 and brought problems of A.R.P. and evacuation, and immunisation ceased. But towards the end of 1940 an intensive campaign began. Posters with a personal and colloquial note were displayed, mothers were spoken to at the child welfare centre, and at school inspection; teachers and members of the corporation were approached; the whole emphasis being on personal discussion and not on formal addresses to groups. A leaflet briefly setting out the case for immunisation, and with a tear-off consent slip attached was distributed to every school child and also to the mothers of pre-school children. Every case admitted to hospital, and more especially every death, was used to point a moral. A public opinion was built up that every careful mother had her children immunised, and that

failure to do so was careless and anti-social. Routine sessions at the clinic for immunisation were reinforced by the Medical Officer of Health going to each school in the town and immunising all for whom consent had been obtained, as many as 150 at a time being treated. No serious reactions occurred, other than a small local abscess in a boy of eight. In that particular case the mother was indignant, and said she had refused to sign a consent; this awkward situation was resolved by the child himself stating that he had in fact forged the signature, and that he wished to have the second injection.

Very few immunisations were carried out by general practitioners, demands for issues of A.P.T. to them being very small. It can therefore be concluded that the figures for immunisations done by the Medical Officer of Health closely approximate to the total done, and although in the past four years there has been some increase in the number of immunisations by general practitioners the records, which are now more complete, show that over 80 per cent. of immunisations in the town are by the public health service. Pre- and post-Schick tests were not carried out, nor were any tests for sensitivity made, as it was considered that there were not sufficient advantages to balance the damage to the scheme which might arise from an excessive number of injections. Table II shows the number of completed immunisations administered by the Medical Officer of Health in each year from 1937 to 1951. The figures are for primary courses only.

TABLE II

Year	Immunisations
1937	883 (single dose)
1938	220 (mostly single dose)
1939	nil
1940	nil
1941	704
1942	817
1943	626
1944	275
1945	315
1946	303
1947	288
1948	389
1949	420
1950	269
1951	369

Booster doses were not given in the earlier years, and while the disease was common this was perhaps not important, as immunity was naturally maintained by repeated exposures to infection, but is now very necessary. The decline of immunity after some years is the explanation of most of the infections in the immunised in the 1951–52 outbreak. An average of about 100 children annually received such booster doses in 1947–51.

In 1952 up to September 443 primary immunisations were done and 987 booster doses were given.

In 1950, and in 1951 up to December, not a single case of diphtheria had been notified. It was estimated that over 90 per cent. of children in the 5–15 year age group and about 70 per cent. in the 1–5 year age group had been immunised.

In December, 1951, there were 15 cases of diphtheria notified, in January, 1952 there were 7, in February 21, in March 32. The outbreak terminated abruptly at the end of March, but since then two single cases have occurred, one on 31st May, and one at the beginning of September.

Source of infection

In the first week of January, 1952, a boy of 5 years developed palatal paresis, and was found to have a positive throat swab. This recalled the case seen some eleven years earlier, and on investigation it was easily found that some six weeks earlier, at the end of November, 1951, this boy had come to the clinic to be placed on the list for tonsillectomy as he had just recovered from a sore throat. The cases in December, 1951, had begun in this boy's class at school, then in a family who had contact with him, and thus infection was carried to another school. In the later phases of the outbreak the linkage was less clear from school group to school group, but in view of the many obvious contacts between children this is to be expected. But it is reasonable to deduce that the boy who was not detected until he developed palatal paresis was the original focus from which the disease spread.

As the number of notifications increased there was some agitation from a few parents and others for school closure, but this was resisted by the Medical Officer of Health, and a forecast was made, and borne out by the event, that the outbreak would end abruptly when it had affected a substantial proportion of the non-immunised population. It was also stated that no wide spread would occur in the immunised.

The effect of immunisation on incidence is interesting. The boy believed to be the origin of the outbreak was not immunised, because his parents specifically objected; after his recovery his parents had immunisation, now probably superfluous, done. Of the 75 cases notified from December, 1951, to March, 1952, 27 claimed to have been immunised, and 48 were certainly not done. It was not possible to confirm the claims of a number among the 27 who alleged that they had been done, but even if all these claims were accepted, and if one also allows for the fact that at least 80 per cent. of the child population are immunised it will readily be seen that the incidence in the non-immunised is at least 7 times as great as in the immunised.

There is little doubt that some of the cases notified were not in fact true cases of diphtheria. As the outbreak continued, and a rising level of suspicion among practitioners in the town led to the taking of swabs more widely in all cases of sore throat and in contacts of notified cases of diphtheria cases were notified as diphtheria which could more accurately be regarded as persons having diphtheria bacilli in nose or throat swabs, and an accompanying clinical condition which was not necessarily diphtheria. It is therefore significant that the proportion of cases of alleged diphtheria in the immunised rose as the outbreak continued. In December, 1951, only 1 case out of 15 claimed to have been immunised, in January, 1952, 2 out of 7, in February 8 out of 21, and in March 16 out of 32. This is exactly what would be expected if in fact transitory carrier states among the immunised were now being detected and notified, a view which was supported by the vagueness or absence of clinical signs in these individuals. A critic of immunisation might suggest that in fact the protection given by immunisation was now failing, but the clinical features, and the abrupt termination of the outbreak, exactly as forecast, negatived this argument. It remains as always true that the diagnosis of diphtheria is a clinical matter, and culture results are only one link in the evidence, whether they are positive or negative. We have all seen undoubted, and even fatal cases of diphtheria with one or more negative cultures, and we can also expect transitory carrier states in the immune after recent exposure to infection.

The age distribution of the cases was:—

1 to 5 years	28
5 to 10 years	34
10 to 15 years	2
Over 15 years	11

Large numbers were brought to the clinic for immunisation as a result of this outbreak, and up to September, 1952, 987 booster doses have been given this year, and 443 doses in primary courses of injections.

No deaths occurred in this outbreak, and the cases were mild. At this time there was no general rise in the incidence of diphtheria in the country, and indeed none in the neighbouring districts.

Summary

The incidence of diphtheria in Darwen from 1937 is reviewed, and an account is given of the progress of immunisation. An outbreak arising from a missed case is described, and the value of immunisation in control is discussed.

NOTIFICATION OF INFECTIOUS DISEASE IN ENGLAND AND WALES, NOVEMBER, 1952

Issued from the General Register Office, Somerset House, W.C.2.

	Nov. 1st	Nov. 8th	Nov. 15th	Nov. 22nd	Nov. 29th	Average weekly figures for November 1951
Scarlet Fever	2,099	1,976	2,065	2,226	2,399	1,312
Whooping Cough	1,456	1,449	1,780	1,854	2,138	1,707
Diphtheria	29	27	44	41	20	39
Measles, excluding Rubella ...	11,229	11,264	12,035	12,134	14,791	2,018
Acute Pneumonia	575	503	452	519	619	471
Meningococcal Infection ...	34	30	29	28	29	39
Acute Poliomyelitis (Paralytic)	65	81	82	62	56	51
„ „ (Non-paralytic)	21	36	22	21	21	19
Ophthalmia Neonatorum ...	38	34	29	35	21	41
Puerperal Pyrexia and Puerperal Sepsis	259	211	226	227	244	236
Dysentery	414	213	211	158	197	207
Paratyphoid	7	2	5	8	4	16
Typhoid	8	4	3	3	1	4
Smallpox	—	—	—	—	—	—

No cases of Cholera or Plague or Typhus Fever.

SECTION II.—PUBLIC HEALTH LABORATORY SERVICE

Issued from the Office of the Medical Research Council, 38, Old Queen Street, Westminster, S.W.1.

A POWDER-DUSTING TEST FOR THE CONTROL OF DISH-WASHING AND THE COMPARISON OF DETERGENTS

F. C. Brookes, County Sanitary Inspector,
West Riding County Council, and

H. Fennell, B.Sc.,
Public Health Laboratory, Wakefield.

In 1950 an attempt was made to improve the washing-up in canteens at twenty West Riding schools by using a bacteriological swabbing technique (Brookes and Burrell, 1950). The experiments were carried out over a period of six months and 600 swabs were examined. Though the results showed that the swab-rinse technique was sufficiently reliable for the control of dish-washing methods, it became abundantly clear that it was too cumbersome and expensive in time and materials to provide a practical test for use in the field. The inevitable time lag between swabbing and acquainting the washing-up staffs with the results tended to slow down and, in some cases, prevent improvement. It was expecting too much of a staff to recollect what had possibly gone wrong on any particular day and to relate the unsatisfactory bacteriological result to their methods of a week ago. It was felt, therefore, that a simple test of dish-washing methods was badly needed—one, moreover, which would present conditions clearly to the workers themselves at the time of testing.

The use of powdered carbon applied with a camel hair brush has been recommended as a field test (Report, 1951) and this seemed to be a suitable starting point. The basis of the test is that grease present *in even very small amounts* will pick up and hold the powder so that it shows as a black or grey mark.

A request in 1951 to compare the merits of four organic detergents proposed for use in West Riding schools provided an excellent opportunity of studying the technique and it was as a result of this study that the present method was finally evolved.

Powder-dusting Technique

Materials

Atomizing powder blower with pneumatic bulb, De Vilbiss powder blower No. 36, supplied by Aerograph Co. Ltd., Lower Sydenham, London, S.E.26.

Large camel hair brushes with a diameter at the ferrule of about ½-inch. The type known as a decorator's camel hair mop, size 6 or 8, is suitable.

Black Powder. 7 parts lamp black, 2 parts pulverized graphite and 1 part pulverized gum acacia. The graphite and gum acacia should be pulverized as finely as possible (Reynolds and Branson, Leeds).

White Powder. "Lanconide" (Barnes and Crompton, Preston).

Method

Utensils are tested when dry after washing and rinsing, any remaining drops of water being carefully blotted with a clean tea-towel to avoid smearing.

Crockery

Black powder is used. With plates, five or six light puffs of powder are blown on to the surface, the nozzle being held two to three inches above the plate; with cups and beakers the powder is blown on to an area approximately two inches deep around the rim. When laid the powder should be scarcely visible; a thicker layer tends to destroy the value of the test for demonstration purposes. The powder is then brushed over the surface with a camel hair mop. Plates which are physically clean brush clean except for powder retained in abrasions on the rim or on badly worn surfaces. The general retention of powder in abrasions on the rim was found to be a useful proof that the powder had been applied.

Cutlery and Glassware

White powder " Lanconide " is used for cutlery and glassware as the black powder does not give the necessary contrast. " Lanconide " is not so effective in the blower as the black powder and application with a brush is necessary.* A little of the powder is taken up on the tip of the brush and distributed by lightly brushing over the appropriate surface of the utensil being tested; this is continued until all excess powder, except that held by grease, is removed. The blades, tines and bowls of knives, forks and spoons, and areas two inches deep around the rims of glass tumblers are examined in this manner.

A Comparative Trial of four Organic Detergents by Powder-dusting

The technique just described was mainly evolved during a comparative trial of four detergents. It is relevant to describe this in some detail so as to indicate some of the pitfalls to be avoided.

As it was essential that the conditions under which the detergents were to be used were as much under control as possible, the work was done at one school only, each detergent being tested for a week in turn. This was considered to be the minimum time in which conclusions could be reached regarding their most effective use, and it allowed each detergent to deal with a complete week's menu.

Swillington County School was selected for the experiments because of the consistently good work of the staff in 1950, when their mean colony counts were 9 per utensil for plates and 38 for cutlery. The school kitchen has one washing-up unit consisting of a 36-inch white glazed stoneware wash-up sink and a metal rinsing sink, with wooden draining boards on either side of the unit. The wash-up sink holds 25 gallons, with a recommended working capacity of 10 gallons. The rinsing sink, which holds two galvanized wire plate racks, is gas-heated to maintain the water at a temperature suitable for sterilization. Two sizes of plate racks are in use holding 7 deep and 12 shallow plates respectively. Approximately 140 school dinners are served on the premises daily.

The washing-up, cleaning-up and laying of tables is done by a staff of four part-time workers, the washing-up being done by two of them on alternate days. None of these workers had been present during the experiments in 1950 and their knowledge of hygienic dish-washing was limited to that passed on to them by the previous part-time workers, who were now engaged full-time in the kitchen.

Each of the four detergents (W, X, Y and Z) was tested in turn, first by using a maximum dose, then by trying to find a dose from which results were

* A white powder consisting of kaolin 7 parts, cornflour 7 parts, and light magnesium carbonate 2 parts, by weight, has since been used in the powder-blower successfully.

unsatisfactory, and finally by establishing an economic optimum dose for each. Ten gallons of water were used for all the washing-up waters which are recorded. The staff were instructed in the proper use of the mixing valve so as to ensure that water was filled into the sink at as near 56° C. as possible. This was done so that the temperature of the water in the sink when filled was 54–55° C. The required dose of detergent was added, stirred up, and the sink filled with scraped plates. The temperature of the water fell to between 52 and 50° C., sufficiently cool for hand-washing to be begun. All the plates were scraped before the washing-up was started, so that a steady flow of dirty plates could be taken through the wash-up sink. The rinsing sink was kept at a sterilizing temperature of between 80 and 90° C. and the plates, in racks, were submerged in it for approximately 30 seconds. After washing and rinsing the plates were allowed to dry in the racks (Mann, 1948).

Plates

In each wash-up a sample plate for powder-dusting was taken from the first rack and from every alternate rack up to and always including the last rack out of that particular wash water. The number of samples obtained ranged from 5 out of 95 plates washed to 16 out of 278, a total of 316 plates being examined by powder-dusting.

There was a little difficulty in regard to the adoption of a suitable standard. It seemed at first that the test was too stringent and that it was too much to expect plates to be quite free from grease. The evaluation of results was also complicated by the fact that water-drop marks and abrasions of the plate surface were also brought up by the powder-dusting. These latter in particular were always more evident when powder was applied with the brush. After a little practice it became comparatively easy to distinguish the smear of a grease mark from rinse-water drop marks. A drop of water evaporating from a plate surface tends to leave a trace of chemicals around its periphery and this shows as an open ring when brushed with powder.

When powder was applied directly with the brush, it was almost impossible to avoid applying it in excess. This entailed brushing for a considerable time to remove the film of powder from the clean portions of the plate surface till the smallest abrasions were made visible, together with water-drop marks and grease.

The method of light dusting has the advantage of leaving a clean-looking surface if there is no grease present, and this is particularly useful when demonstrating. Comparisons were made over a large number of plates which were first lightly blown and then heavily brushed. No additional grease was found in this way. Rinse-water drop marks and abrasions are not significant in themselves and, if shown, have to be discounted in assessing the cleanliness of the plate. Consequently there is a practical disadvantage in powdering the plate too heavily. A light powder-dusting with the powder blower having been adopted as standard practice, it was soon found in use that this gave very sensitive control of dish-washing methods. It quickly demonstrated where plates had been held for washing without having been turned round and where perfunctory washing had left grease smears, particularly on the rims and concave surfaces of the plate bowls. It was found that *one wipe of the dish-cloth* over the whole surface was sufficient to provide a plate free from grease, provided that sufficient detergent was being used. It was noticed that the workers had a tendency to wipe each plate very quickly probably half a dozen times, the cloth being flicked around the plate. This was altered to less but more careful wiping, which, though taking no more time, resulted in an immediate improvement in the condition of the washed plates. The staff were

very consistent in their working methods and, having made the improvement in the first week, maintained it throughout the experiment without difficulty.

It was then realized that the problem of establishing a standard had resolved itself. Little difficulty was experienced, so far as the staff were concerned, in providing washed plates which, on being powder-tested, gave a completely negative result without a trace of grease, either back or front.

As a control during the second fortnight, when most of the plates tested at Swillington School were found so completely satisfactory, surprise visits were paid to other schools, where from 8 to 12 plates selected at random were subjected to the powder-dusting test. The same technique was used and the plates, generally, were found to be very unsatisfactory. At one school which was visited twice, an improvement was noted on the second visit.

Having established consistent working conditions, which continued testing showed to be reliable, it was then possible to note what happened when the detergent dose was insufficient. It was found that some abrasion marks came up more significantly under the light dusting and that small pitted depressions on the surface retained small smears of grease giving the plate a " measled " appearance. This effect was noted on even the first plates washed in such a solution, and the subsequent plates tested were found to be similar.

We now had at our disposal a method of checking whether washed plates were free from grease and, by using judgment, it was possible to distinguish between the lack of sufficient detergent and ineffective methods of washing.

Cutlery

Greater difficulty was experienced in dealing with the cutlery than with the plates. Eventually a method of washing was found which gave satisfactory results and which interfered less with the routine than a previous recommendation had done. Hot water (55–60° C.) suitably dosed with detergent was provided for the reception of cutlery immediately after use, about 3 gallons being used in a 5-gallon container. This was sufficient to deal with all the cutlery used at 140 meals. After the plates had been washed, the cutlery was transferred to the wash-up sink, well stirred about and placed in the cutlery baskets which were then immersed in the rinsing sink for at least two minutes. No attempt was made to insist on the individual wiping of each utensil with a dish cloth, but it was pointed out that every reasonable opportunity of agitating the water or disturbing the utensils should be taken, as this would tend to assist grease removal. The cutlery was soaked for at least an hour before being washed, and, after rinsing, was dried off with clean tea towels.

When powder-dusting the cutlery, there appeared at first to be a tendency for the fine abrasions on the surface, particularly on the forks, to hold the white powder, but when prolonged soaking was used, results were finally obtained in which all the utensils examined brushed clean. Some confusion was caused through inadvertently brushing some of the utensils which were slightly damp, thus producing a " whitewash " smear which was at first thought to be grease. Ordinary droplet marks on cutlery were found to brush away almost completely.

Results

Plates

Various methods of recording the results were tried. It was only really necessary in assessing the performance of detergents to adopt a plain standard of " satisfactory " when a plate was completely free from grease and " unsatisfactory " when it was not, but it was evident that some method of grading was advisable when controlling the work done by dish-washers. We had to

distinguish in this latter case between bad work and a " near miss " and so on. Finally, a numerical scale from 10 downwards was adopted, 10 signifying that the plate was free from grease back and front, or with not more than about ¼-inch square of grease on the rim or the back of the plate, the bowl being clean. Any deviation from this was marked 9 if the amount of grease was slight (say not more than ½-inch square), 8 if there were more and so on. The estimation of the amount of grease was done by eye as actual measurement was impracticable. The distribution of the grease marks was also taken into account, a lower marking being given for a wide distribution.

TABLE I

Comparison of Four Detergents by Powder-Dusting

Code No. of wash	Dose (oz. per 10 gallons of water)	Time wash water in use (mins.)	Temperature °C.			No. of plates tested	Powder-dusting result	
			Wash begun	Wash finished	Rinse sink		Aggregate score*	Mean
W. 1	4	18	54	39	90	7	52	7·4
2	3	25	61	43	90	9	85	9·4
3	2	11	55	43	85	5	36	7·2
4	3	23	50	39	85	8	72	9·0
5	3	23	55	39	89	7	64	9·1
6	3	23	52	39	88	7	66	9·4
7	3	23	54	38	90	8	76	9·5
X. 1	3	25	49	37	90	9	88	9·8
2	2	17	55	39	89	6	57	9·5
3	3	20	55	42	89	9	90	10·0
4	2½	26	51	39	84	9	87	9·7
5	3	35	55	35	85	12	119	9·9
6	2½	33	48	36	—	8	78	9·7
7	2½	26	55	42	—	8	80	10·0
8	3	30	54	38	92	10	100	10·0
Y. 1	3	35	54	36	87	15	150	10·0
2	2¼	30	57	36	—	16	159	9·9
3	1½	22	50	38	82	8	72	9·0
4	3	34	52	37	82	9	90	10·0
5	3	29	55	40	85	12	120	10·0
Z. 1	5	38	50	35	82	15	150	10·0
2	3	20	55	37	81	9	89	9·9
3	4	34	50	36	90	13	130	10·0
4	4	45	54	34	87	15	150	10·0
5	4	43	55	35	90	14	140	10·0

* No grease present or not more than about ¼-inch square—10 marks. Not more than about ½-inch square—9 marks, etc.

Table I gives an analysis of the results from 25 washings, the code letters in the first column referring to the four detergents used. The washings recorded, with two exceptions, dealt with plates only, so as to allow for comparision of time, temperature and work done. In the excepted washings (Y4 and Z4) the plates were finished before the detergent was used up and additional work was done.

Only mean results of 10·0 were considered to be satisfactory when assessing the merits of the different detergents. On this basis it will be seen from the table that the optimum doses for Detergents X, Y and Z were 3 oz., 3 oz.

and 4 oz. respectively, all of which were found to give entirely satisfactory powder-dusting results at temperatures varying from 35° to 55°C. and for times up to 45 minutes with Detergent Z. The cost per dose was found to be 1·6, 2·9 and 1·0 pence respectively.

The results for Detergent W were unsatisfactory partly because the test was not standardized, and partly because the staff had not then reached a sufficiently high standard of washing-up. A gradual improvement is shown in washings W 4, 5, 6 and 7. This detergent, however, was found to be unpleasant to use, and from the results it was unlikely that a 3 oz. dose would be sufficient. A bigger dose than this would have been uneconomical and it was felt that it would be a waste of time to continue testing.

Cutlery

Owing to the time taken to establish the fact that cutlery could be washed sufficiently well to satisfy the powder-dusting test, it is not possible to give comparative results of the cutlery examinations. No special marking scale was used; a utensil brushing clean was classed as "satisfactory" and all the others "unsatisfactory." About 450 cutlery utensils were tested and the 96 examined on the last four days were all satisfactory.

The cutlery was apparently best dealt with by Detergent Z, followed closely by X. Detergent Y was effective, but there was yellow staining of worn cutlery.

General

It was found generally that a given volume of water needed a certain amount of detergent to obtain effective grease removal. Below this minimum unsatisfactory results in the form of "measled" plates were obtained, even at the beginning of a washing.

The temperature of the water within the limits tested, 55——34°C., did not affect the efficiency of the detergents. Fairly good results were obtained on one occasion with Detergent Z when a few plates were washed in cold water (17°C.).

No difficulty was found during the last three weeks in using a sufficiently concentrated solution for at least 30 minutes, and on occasions for over 40 minutes. Unless, however, the water is as hot as possible when washing is begun, it becomes uncomfortably cold and is thrown away unnecessarily soon. It was noticed that the workers tended to discard water at 39°C., but when they realised that satisfactory results were being obtained down to 34°C. they continued to use the same water.

The menus served throughout the investigation were recorded but we did not find any connexion between an estimate of the "greasiness" of the respective meals and the efficiency of the dish-washing.

Correlation of Powder-Dusting and Bacteriological Examinations

It became clear to us during the early stages of this investigation that the powder-dusting test was likely to give exceptionally good control of dish-washing operations. It was simple and, what was very important, results were available immediately and in a form to be readily appreciated by the workers. The question arose as to how the test would compare with bacteriological examinations using the swab-rinse technique, and whether it could possibly replace this as a control of dish-washing methods.

Bearing in mind that films of grease hold bacteria on to soiled surfaces and prevent effective sterilization either by hot water or chemicals, it would seem that a test which visually demonstrates the absence of grease could be used

to infer a satisfactory bacteriological standard of cleanliness, provided it is known that sterilization is being effectively done. Where heated rinsing sinks are in use it is comparatively simple to ensure that the conditions for sterilization are met. It was decided to try to find out whether, when such a sink was being properly used, there was a good correlation between the results of the bacteriological examination of utensils by swabbing and testing by powder-dusting.

During the bacteriological examination of dish-washing methods carried out in 1950 it was noted that, although considerable care was taken to ensure that the heated rinsing sinks were properly used, some high colony counts were obtained which it was felt could only be explained on the assumption that the actual washing process had not been efficient.

Sample swabs for comparison with the powder-dusting results were collected on four separate days. On each day eight swabs from 32 plates, out of approximately 280 washed, were taken. As it was not advisable to powder-dust and then swab the same plates, the following method was adopted. A single plate was selected from every alternate rack for powder-dusting, and the plates on either side of it (*i.e.* the plates which had been washed immediately before and after) were put on one side for later swabbing. These latter were then combined in fours and swabbed, so that each bacteriological sample compared with two powder-dusted samples. Swab No. 1 was taken from the two plates from each of racks 1 and 3 on either side of the plates selected for powder-dusting and so on.

A similar control was not easy to arrange for the cutlery, and for this four articles were selected at random, swabbed, allowed to dry and then powder-dusted. The results of the powder-dusting were classified as " satisfactory " if the articles brushed clean, and " unsatisfactory " if there was more than a minimum of marking. Twenty-one swabs from cutlery were taken on the same four days as those from the plates.

The swab-rinse technique used for the examinations was a modification of that recommended for use in the United States (Report, 1943). Yeastrel agar was used in place of tryptone glucose extract agar and the buffered distilled water was distributed in 10 ml. amounts, 2·5 ml. being plated and incubated for 48 hours at 37°C. (Burrell, 1950). The bacteriological standard adopted as satisfactory was that the average colony count per utensil should not exceed 100 colonies.

Results of Powder-Dusting Correlation Test

The results of the comparative examinations are given in Tables II and III. All the mean results shown in Table II attained the maximum of 10·0 with the

TABLE II

Comparison of Powder-Dusting with Bacterial Counts

PLATES

Detergent used	Temperature of wash water °C.	Rinse water temp. °C.	Time of wash (min.)	No. of plates washed	Powder-dusting		Bacterial Colony Counts 48 hr. at 37°C.			
					Plates tested	Mean result	No. of Swabs	Min.	Max.	Mean
X ...	55–35	85	35	225	12	9·9	6	0	5	2
	54–?	—	8	49	3	10·0	2	0	4	2
Y ...	55–40	85	29	187	12	10·0	6	3	41	12
	54–?	—	?	79	4	10·0	2	3	7	5
Z_1 ...	50–35	82	38	267	15	10·0	8	0	8	3
Z_2 ...	54–34	87	40	267	15	10·0	8*	0	26	9

* One count spoiled by spreading organism.

exception of the first, which was reduced to 9·9 by the presence of a little grease on one plate. The corresponding colony counts of the bacteriological examinations are shown in the adjacent columns. One plate count was spoiled by a spreading organism and the remaining 31 colony counts ranged from 0 to 41, with a mean count of 6; 27 of the plates had counts of 10 or less.

TABLE III

Comparison of Powder-Dusting with Bacterial Counts

CUTLERY

Detergent used	Powder-Dusting Results			Bacteriological Results				
	Satisfactory	Unsatisfactory	Satisfactory	No. of Swabs	Colony Counts 48 hr. at 37°C.			
					Min.	Max.	Mean	Satisfactory
			Per cent.					Per cent.
X ...	22	2	91·3	6	0	18	6	100·0
Y ...	8	4	66·7	3	3	147	54	66·7
Z_1 ...	20	4	83·3	6	4	16	8	100·0
Z_2 ...	24	0	100·0	6	5	42	17	100·0
Totals	74	10	88·1	21	0	147	17	95·2

Comparing the results on the cutlery given in Table III, it will be noticed that only on the last occasion (Z_2) was the cutlery given full marks on the powder-dusting tests. The corresponding colony counts are again shown in adjacent columns. There was one count above 100 for forks with Detergent Y and these had been evaluated under the powder-dusting test as only 50 per cent. satisfactory. The remaining 20 samples had colony counts ranging from 0 to 42, with a mean count of 10. There were 14 counts of 10 or less.

Washing without Sterilizing Rinse

It was then decided to find out the bacteriological condition of plates whose physical cleanliness had been determined by the powder-dusting test. The plates were washed in Detergent X solution (3 oz. to 10 gallons of water) at 55–35°C. and then rinsed in a bath of clean water at 55–40°C. A plate was selected from each rack of rinsed plates for powder-dusting and adjacent plates were taken for bacteriological swabbing. About 10 per cent. of the plates washed were powder-dusted and 40 per cent. were swabbed. The results obtained are set out in Table IV. For the purpose of this experiment the marking system for powder-dusting was made more sensitive by confining a score of 10 to plates without grease. Plates showing traces of grease insufficient to justify marking down to 9 were marked at $9\frac{1}{2}$.

The results of these experiments, made in March 1952, showed that the standard of washing-up had been well maintained by the staff. A little falling off was apparent in the results of the powder-dusting test, but on the whole they indicated that grease removal was comparatively effective. The colony counts were very low, only 4 out of 31 being unsatisfactory. The highest count was 275 and the mean 51.

TABLE IV

Washing Without a Sterilizing Rinse

Day	Powder-Dusting Results			Bacteriological Examination Results				
	Number of plates tested	Aggregate† score	Mean	No. of swabs examined	Unsatisfactory*	Colony Counts 48 hr. at 37°C.		
						Min.	Max.	Mean
1st ...	24	237½	9·9	13	—	5	65	21
2nd	30	291½	9·7	9	3	8	275	102
3rd ...	18	174½	9·7	9	1	7	120	43
Total	72	703½	9·8	31	4	5	275	51

* Colony counts of more than 100 per utensil are considered unsatisfactory.

† No grease present ... ... 10 marks
Not more than about ¼-inch square ... ... 9½ marks
Not more than about ½-inch square ... ... 9 marks etc.

On the third of these experiments a further series of 9 swabs was taken after the plates had been put through the sterilizing sink. Comparative results for powder-dusting and both series of swabs are given in full in Table V.

TABLE V

Results after Washing With and Without a Sterilizing Rinse

Rack No.	Powder-Dusting Result	Colony Counts 48 hr. at 37°C.	
		Without sterilizing	After sterilizing
1	9	—	—
2	10	15	1
3	10	—	—
4	10	7	17
5	10	—	—
6	9½	24	41
7	9½	—	—
8	10	31	58
9	9½	—	—
10	9½	23	32
11	9½	—	—
12	9½	48	41
13	9½	—	—
14	9½	120	440
15	10	—	—
16	10	54	41
17	9½	—	—
18	10	68	109
Means	9·7	43	87

The dusting scores are recorded as in Table IV.

Beaker-Washing

In November, 1951, a question arose as to the best way of washing plastic beakers after use for drinking water. Three methods were tested by powder-dusting and bacteriologically.

(A) The beakers were put in hot water and detergent in the washing sink, rinsed, and packed in cutlery baskets for immersion in the heated rinsing sink.

(B) The beakers were packed in cutlery baskets, immersed in hot water and detergent in the washing sink and shaken about for 15 seconds before being transferred to the heated rinsing sink.

(C) The beakers were washed in hot water and detergent in the washing sink, and individually wiped with a clean dishcloth before being put in the cutlery basket for transfer to the heated rinsing sink.

In all the methods, hot water at 50°C. was used for washing, and the water in the rinsing sink was maintained at a temperature between 74 and 84°C. Detergent X of the previous experiments was used (3 oz. to 10 gallons of water). The cutlery baskets were rectangular open mesh galvanized wire baskets about four inches deep, with a wire mesh lid which could be fastened. Twenty beakers were laid on their sides in each basket, with precautions against accidental nesting during immersion in the rinsing sink, which was for at least 30 seconds. After rinsing, the beakers were inverted on clean metal trays and allowed to dry. It was found, however, that drying had to be finished with clean tea towels as the beakers did not retain sufficient heat to evaporate the larger drops of rinse water left on the surface.

The washed beakers were examined on four different days by both tests, and a summary of the results appears in Table VI. The results were not "scored" but taken as "satisfactory" when clean, and "unsatisfactory" if grease or film were present. On the first day Method A gave such bad results on the powder-dusting test that the method was discontinued even before the bacteriological results were known. As will be seen from the table, these fully confirmed this judgment. On the remaining three days only Methods B and C were used.

TABLE VI

BEAKER WASHING

Method	Number of powder dustings and results			Number of Bacteriological Examinations and results					
	Satisfactory	Unsatisfactory	Satisfactory	Satisfactory	Unsatisfactory	Colony Counts 48 hrs. at 37°C.			Satisfactory
						Min.	Max.	Mean	
			Per cent.						Per cent.
A. ...	—	4	0	1	2	53	800	450	33
B. ...	21	9	70	15	—	0	26	8	100
C. ...	30	—	100	16	—	0	19	6	100
Totals	51	13	80	32	2	0	800	46	94

With Method B the mean colony count of the 15 samples examined, each consisting of 4 beakers, was 8, and the counts varied from 0 to 26. With Method C the mean colony count was 6 for 16 samples and the counts varied from 0 to 19. There is no significant difference between these results, all of which are satisfactory, i.e., with counts of less than 100.

The powder-dusting test showed that Method B gave only 70 per cent. satisfactory results compared with 100 per cent. satisfactory with Method C, a difference which appears to have some significance.

On three occasions during this beaker-washing test, spot checks were made on the remainder of the washed utensils. Fifty-two utensils were tested bp powder-dusting; 40 were found satisfactory, 11 doubtful and 1 unsatisfactory. Thirteen swabs, from four utensils each, were examined bacteriologically and all were satisfactory. The mean colony count was 12 per utensil, the maximum count being 29.

These results indicate that powder-dusting is a more sensitive test of washing-up methods than bacteriological examination by the swab-rinse technique.

Discussion

The experience of one of us (F. C. B.) in the supervision of dish-washing methods in school canteens has shown that control by bacteriological examination is impracticable for routine use. Expression of results in definite figures gives a misleading idea of their accuracy, the experimental error in plate counts being of the order of plus or minus 90 per cent. To effect an improvement in methods sampling must be maintained over a fairly long period, and the response to poor results is by no means always immediate or readily apparent.

It is doubtful what part, if any, unhygienic washing-up plays in the incidence of food poisoning, but the psychological effect of cleanliness in any one part of the kitchen tends to greater care in other parts. The bacteriological examination of washed utensils in the way referred to above is not really concerned with the type of bacteria present but uses the degree of contamination as a measure of effective washing-up.

Powder-dusting tackles the problem at the point of greatest variability, the actual washing of utensils in the sink. It would appear to be a sufficiently reliable index of the cleanliness of washed utensils for practical use, and has several advantages over any method of bacteriological control. It is cheap and easy to apply. It will demonstrate the results of faulty methods in a way readily appreciable by the workers concerned whilst work is actually in progress. The test is very sensitive and will distinguish between small differences in the quality of dish-washing.

Insufficient attention has been paid in the past to physical as opposed to bacteriological criteria of cleanliness. With plates and other crockery it would appear from our experiments that, if physical cleanliness (*i.e.* freedom from grease and film) can be assured by the use of a test such as this, there is no need for great stress to be laid on sterilization save as a second line of defence. It is advocated, however, that the hot water rinse should be maintained, so as to avoid the necessity of drying with cloths.

The test can be used while washing-up is being done or at any time before utensils are re-used. A larger sample of utensils can be examined than would be reasonably possible with the swab-rinse technique and a more reliable picture of the effectiveness of a wash-up is formed. It is suggested that at least 10 plates, for instance, should be sampled from each wash-up, the plates being selected at regular intervals if the work is in progress and taken at random from the stacked plates otherwise.

At first sight the test may appear clumsy and the method of recording results subjective, depending overmuch on the judgment of the observer and liable to a large personal bias. It must be remembered, however, that the real standard is a complete absence of grease and film and this is not difficult to recognize and obtain with a co-operative staff. There seems to be little point in adopting

a lower standard. For comparative purposes during the investigation it was necessary to adopt the described scale of marking, and we tried to maintain a reasonable consistency in using it. There is no objection to the use by other observers of a scale differing from this so long as the ultimate criterion of judgment remains the complete freedom from grease and film.

Although the test has so far been used only for school canteen kitchens it is obvious that it should prove equally useful in all kitchens in which washing-up is done. Preliminary tests have shown that the white powder will effectively show the presence of grease and mucous film on drinking glasses, and this will permit testing of glasses in public-houses, if they are dry. It is realized, of course, that this latter proviso introduces a practical difficulty in applying the test.

Summary

The swab-rinse technique, though sufficiently reliable for the control of dish-washing methods, is too cumbersome and expensive in time and material for practical work in the field.

A technique of powder-dusting is described which gives better control of dish-washing and a more sensitive differentiation of the performance of detergents than would be possible bacteriologically.

Washed utensils which satisfied the powder-dusting test produced mean colony counts well within the bacteriological standard of not more than 100 colonies per utensil suggested by the United States authorities (Report, 1943), whether or not the washing was subsequently followed by hot water sterilization.

The test is cheap and easy to apply, and shows the results of faulty methods in a way readily appreciable by the workers while work is in progress.

We wish to thank Dr. H. T. Findlay, Director of the Public Health Laboratory, Wakefield, and Dr. J. Wood-Wilson, Deputy County Medical Officer of the West Riding County Council, for the opportunity of carrying out this study and for their helpful advice and encouragement. We are grateful to Mr. F. Hanson, School Meals Officer, and Miss I. Dods, Senior Organiser of School Meals, for their willing co-operation and to the staff at Swillington School for the interest they took in the investigation; to the West Riding Constabulary for the loan of materials, and Detective Superintendent G. Maclean, Glasgow, for the formula of the black powder.

References

Brookes, F. C., and Burrell, J. (1950) Report to West Riding Education Committee (unpublished).

Burrell, J. (1950) *Pers. Comm.*

Mann, B. (1948) *Publ. Hlth., Lond.*, **61**, 82.

Report (1943) *Publ. Hlth. Bull., Wash.*, No. 280.

Report (1951) " Hygiene in catering establishments. Report of the Catering Trade Working Party." *Min. Food, Lond.*

INDEX TO VOLUME 11, 1952

Editorial Matter for

I.—The GENERAL SECTION to
Editor,
Ministry of Health,
Savile Row (Room 207),
London, W.1.
Tel.: REGENT 8411. Extn. 91.

II.—The LABORATORY SECTION
Editor,
Medical Research Council,
38 Old Queen Street,
Westminster, S.W.1.
Tel.: WHITEHALL 4884.

The November Bulletin was issued on 21st November.

Note.

Reprints of some papers are available. A correspondent who asks for a back number should therefore quote the title of the paper desired in case the number is out of print.

(17704) Wt. 8221—7283 3300 12/52 D.L./HE/4